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ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS

REPORT OF A CASE

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It is recognized, as has been stated by several authors, that the actual etiology of true essential progressive atrophy of the iris has not been discovered. The object of this paper is to report another case of a patient with this condition, who has been under observation for four years.

Comparatively few cases of essential atrophy of the iris have been reported, and knowledge of the disease is therefore incomplete. It is possible that some of the earlier cases reported may have belonged to the group of atrophies of the iris caused by, according to Fuchs,[1] (1) long-continued or recurring inflammation, (2) increase of tension involving the blood vessels at the root of the iris, (3) traction on the iris or iridodialysis, (4) too thorough absorption of swelling lenticular material or (5) advanced age or an unrecognizable cause. This was noted by Barr,[2] in 1934, who further stated that essential atrophy is progressive, so that congenital anomalies, such as aniridia or colobomas, should be easily ruled out.

REPORT OF CASE

Miss A. C., a graduate nurse aged 29, was first seen in September 1933, when she was referred for examination of her eyes. She stated that her vision was normal but that she wished advice concerning the vertical elongation of her left pupil. This phenomenon was first noticed by her dentist in June. At that time her eyes were examined, and glasses were prescribed, which were discarded within two weeks.

A thorough physical examination by her family physician revealed no pathologic condition except an absence of the knee jerk reflex. This, however, had been known for several years, as life insurance had been refused on that one count. Her physical examination had included Wassermann tests of the blood and spinal fluid, both of which were reported negative. She denied having had a serious illness or a recent injury. She was a very healthy-appearing person who weighed

1. Fuchs, E.: Text-Book of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, p. 668.

2. Barr, Albert S.: Essential (Progressive) Atrophy of the Iris: Report of a Case, Arch. Ophth. **12**:567 (Oct.) 1934.

140 pounds (63.5 Kg.) and was physically active, engaging in such sports as swimming, tennis, horseback riding and, occasionally, golf. There was a history of an injury in 1920. The patient fell when racing on ice skates, striking her forehead and being knocked unconscious. She was told of a slight concussion, but recovery was complete in a few days, and it is difficult to believe that the accident so many years previously would have any connection with this ocular condition.

Ophthalmic examination showed vision of the right eye to be 20/20 + and that of the left 20/20—. The right eye appeared normal. It was quiet; the iris was light bluish green, and the pupil was round and active. The left eye was quiet, and the color was the same as that of the right, but the pupil was not round. There was a vertical angle superiorly, as shown in *A* of the figure. The pupil had the shape of a hanging drop. The slit lamp study showed no evidence of inflammation. The fields and blindspot of each eye were plotted and were normal. Examination carried out with the pupils under the influence of a cycloplegic revealed practically no refractive error. The tension, recorded with a new Schiötz tonometer, was 20 mm. in each eye. The fundi were normal.

On November 20, or nearly three months later, the patient was seen again. Her pupil was greatly elongated vertically and pulled slightly temporalward (inset in the figure). The patient reported for observation every three or four months.

In February 1935 a drawing was made which depicted some real progressive atrophy of the iris (*B* of the figure). The fields were normal, and the tension was 15 mm. for the right eye and 20 mm. for the left. The slit lamp showed the stroma of the iris to be drawn upward and temporalward. Between 12 and 2 o'clock only the pigment layer of iris was left, and in some parts of the irregular pupillary area there was a uveal ectropion.

In November 1935 there had been more extensive atrophy and disappearance of stroma of the iris, the barren area extending from 12 to 2 o'clock and toward 3 o'clock. The tension and the fields had remained normal. There had also been a disappearance of the pupillary area of pigment layer in the upper temporal quadrant (*C* of the figure).

In February 1937, nearly four years after the onset of the disease, the first anterior synechiae were discovered by use of the corneal microscope. The pupillary area of the iris below and nasally seemed to be thinner and tense, and the iris was adherent to the corneal endothelium in an area below, about 2 mm. from the limbus and extending 4 mm. horizontally, and one adhesion was seen superiorly, toward 11 o'clock and about 2.5 mm. from the limbus (*D* of the figure). Dr. M. U. Troncoso made a gonioscopic examination at this time and reported as follows:

"The iris has a faded gray color, and its ridges look more distinct than in the normal eye. The nasal border of the coloboma shows distinctly the normal uveal rim, while in the temporal, and especially in the lower, part, the border of the iris has been split in its two layers. The retinal layer is in place and shows a peculiar meridional striation. The stroma layer is atrophic and has receded downward, leaving a wide, triangular area exposed. The examination of the angle of the anterior chamber at this place showed a wide anterior peripheral synechia (*E* of the figure), which is attached to the limbus rather forward, almost to the transparent edge of the cornea. This synechia is partial. On each side the angle is open, the brown band of the ciliary body being clearly observed. The canal of Schlemm is not apparent and merges with the white sclera. In the upper part of the angle the coloboma does not reach the scleral limbus. A narrow stump

of the retinal layer of the iris remains. Behind it the ciliary processes appear and show no evidence of inflammatory disease. On the nasal side of the coloboma there is also a narrow anterior peripheral synechia. Except for these synechiae the angle is open all around the limbus."

In none of the reported cases of this disease has gonioscopy been used and the angle of the anterior chamber studied during the life of the eye. The discovery of these peripheral synechiae may be important from the standpoint of pathogenesis. In a majority of the cases the synechiae were reported after histologic examination of the enucleated eye.

COMMENT

The first cases of this unusual disease were reported in Europe in the last decade of the nineteenth century. The first clearcut clinical description of this condition was written in 1903 by Harms.[3] In his group he included G. Lindsay Johnson's [4] case of progressive atrophy of the iris, reported in 1886, and Carl Hess' [5] case of "non-traumatic iridodialysis," the report of which appeared in 1892. The earliest case of this condition described in the American medical literature is that reported by Casey Wood [6] in 1908, who described his case in detail in 1910 at the meeting of the Section on Ophthalmology of the American Academy of Ophthalmology and Otolaryngology. Among the earlier cases were those reported by Zentmayer [7] in 1918, de Schweinitz [8] in 1915 and Lane [9] in 1917.

Fine and Barkan [10] in June 1933 saw a 9 year old patient who had had changes in the iris of each eye for over four years. These were noticed by the family before the child was 5 years old. The authors' descriptions of the early increase in the tension of each eye, of the ineffective operative treatment and of the thinning of the sclera, which became bluish, promptly places this condition in the class of buphthlmos. It was evidently congenital and progressive and will terminate as true buphthalmos always does. It does not earn a place among the true essential progressive atrophies of the iris.

The eccentric position of the pupil is the earliest symptom of essential progressive atrophy of the iris. This sign has been particularly stressed by illustrations in the reports of the case histories. The irregular enlargement of the pupil has always been described, and several authors

3. Harms, C.: Klin. Monatsbl. f. Augenh. **41**:522, 1903.
4. Johnson, G. Lindsay: Ophth. Rev. **5**:57, 1886.
5. Hess, Carl: Klin. Monatsbl. f. Augenh. **30**:103, 1892.
6. Wood, Casey: Ophthalmoscope **8**:858, 1910.
7. Zentmayer, William: Am. J. Ophth. **1**:510, 1918.
8. de Schweinitz, George E.: Tr. Am. Ophth. Soc. **14**:250, 1915.
9. Lane, L. A.: Ophth. Rec. **26**:285, 1917.
10. Fine, Max, and Barkan, Hans: Am. J. Ophth. **20**:277, 1937.

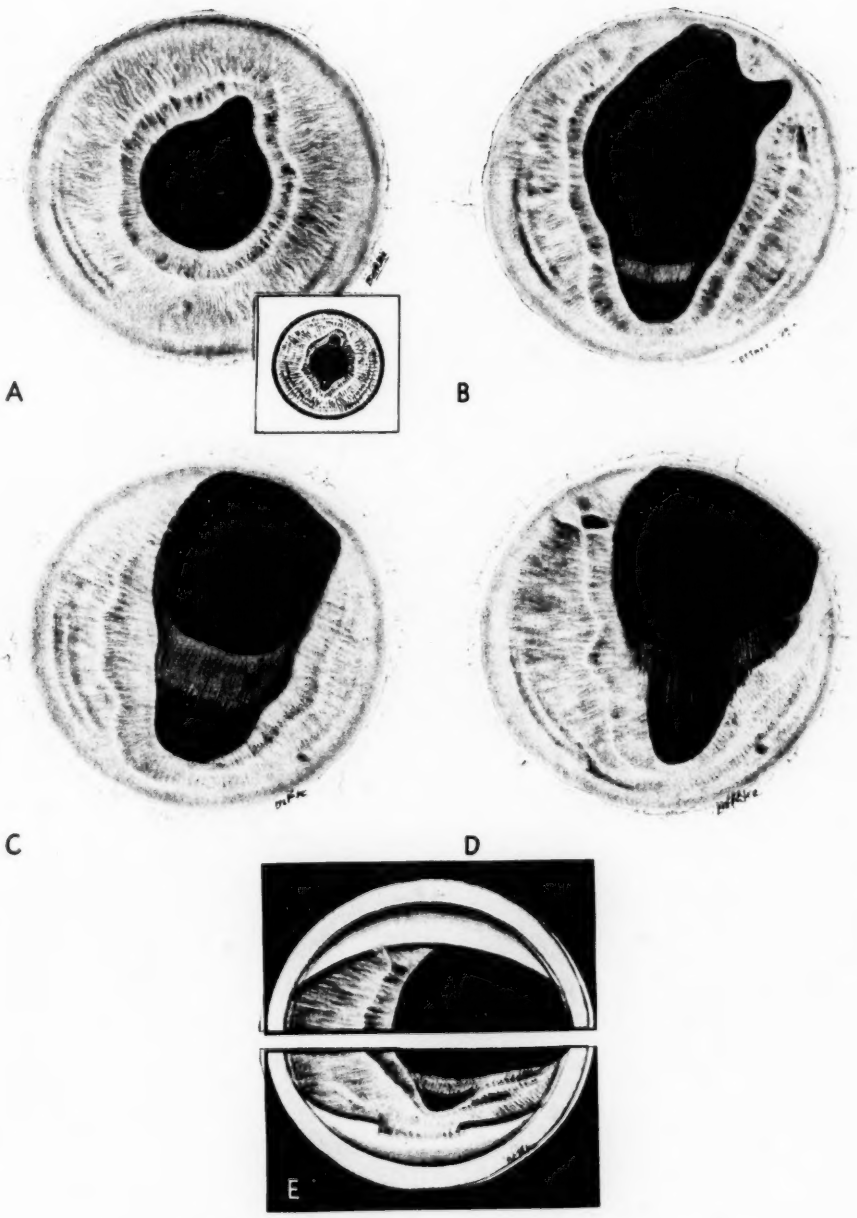
have offered explanations of these changes. Rochat suggested that the change in the size and position of the pupil may be "the result of a circumscribed shrinking of the iris by some pathological process" or "by assuming a primary atrophy of the iris, in which case the pupil would naturally be drawn to the side where the iris is less atrophic." This explanation may well be applied to the condition in my case. The pupil, which was first vertically elongated upward, was later displaced downward, being dragged by the less atrophic iris, before the progressive atrophy advanced into the upper temporal quadrant.

It is interesting to note in the study of the records of cases how few cases were studied before the onset of the increase in the tension and other symptoms of glaucoma. De Schweinitz' patient was under observation for two years before any sign of glaucoma appeared. Rochat and Mulder's [11] patient was seen in 1917, when the atrophy of the iris was well advanced and the eye was entirely free from any sign of glaucoma. These authors could not report the date of appearance of the signs of glaucoma, as the patient was not seen for six years. When the patient did report, the eye was blind and in a state of absolute glaucoma. My patient has been seen fairly regularly, and accurate observations have been made. Her vision is 20/20, and her fields and fundus are normal. The fact that her age was 29 at the onset makes the condition fit very well in de Schweinitz' classification as to age and sex. He listed seven females and five males, and the ages of the females ranged from 20 to 47.

Although it has been suggested that some constitutional fault might be an etiologic factor in this unusual disease, each case report has stated that the general physical examination has usually resulted in negative findings.

In all the reported cases the disease has been unilateral. Feingold [12] expressed the belief that in his case there may have been congenital vascular disturbances near the lesser vascular circle, which could have induced changes in the iris. Lane stated the opinion that a disturbance of circulation due to sclerosis of the vessels of the iris might be a causal factor. Lane's patient had latent tuberculosis. Wood's patient was reported to have had fibrinoplastic iridocyclitis, and it is possible that the condition was not true essential atrophy, as low grade uveitis is not accepted as a causal factor. De Schweinitz reported that his patient had had an attack of retrobulbar neuritis of the opposite eye two years previously and that "although it might be the forerunner of a disseminated sclerosis, it could hardly be related to the iris lesions." Zentmayer suggested that sclerosis of the vessels of the iris cause loss

11. Rochat, G. F., and Mulder, W.: Brit. J. Ophth. **8**:362, 1924.
12. Feingold, M.: Am. J. Ophth. **1**:1, 1918.



Appearance of the eye. *A* shows the appearance in September 1933, and the inset shows the appearance on November 20. *B* shows the condition as seen on Feb. 19, 1935; *C*, the appearance on Nov. 19, 1935; *D*, that on March 4, 1937, and *E*, the gonioscopic view on March 4, 1937.

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of nutrition, which might create atrophy of the stroma, but he did not advance a theory for the cause of this vascular sclerosis. In de Schweinitz' first paper in 1915 it was stated that "a toxin liberated by some constitutional or local infection may be responsible for exciting iris-vascular disease, whereby nutrition is lowered and atrophy follows."

The treatment of the disease has been very unsatisfactory. In a majority of the cases in which treatment with miotics or surgical intervention has apparently been successful, the results have been only temporary. One reluctantly, yet assuredly, accepts the prognosis that in the long run the eye will be lost because of absolute glaucoma. This thought, however, is somewhat overshadowed by the fact that the disease has never affected both eyes of the same patient.

PREVENTION AND TREATMENT OF KERATITIS NEUROPARALYTICA BY CLOSURE OF THE LACRIMAL CANALICULI

REPORT OF A CASE

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The present methods of treatment of neuroparalytic keratitis, though effective, are cumbersome. They interfere with vision and are a nuisance to the patient. Reduction in the secretion of tears is a recognized feature in these cases. Because of the inadequate supply of tears, evaporation leads to dryness and corneal damage. Any measure preventing drying leads to healing. Verhoeff [1] kept the affected eye moist with Ringer's solution, and the corneal epithelium was rapidly restored. Suturing the lids reduces evaporation of tears, and corneal lesions clear. The use of castor oil or liquid or solid petrolatum serves the same purpose, as does also Buller's shield [2] or special protective goggles.

Our studies have convinced us that although the cornea is anesthetic the diminished secretion of tears is the primary factor in the evolution of these corneal lesions. Blocking the canaliculi prevents the escape of tears into the lacrimal sac and should help to prevent drying. In one case of neuroparalytic keratitis this was done, and the corneal lesion healed promptly.

REPORT OF CASE

H. M., an intelligent woman aged 35 years, showed neuroparalytic keratitis in the anesthetic eye six months after the posterior root of the right fifth cranial nerve was cut for intractable trigeminal neuralgia, through a suboccipital approach by craniotomy. When she noticed after operation that emotional tearing was greatly diminished in this eye she observed the eye more closely from day to day and became convinced that it was constantly less moist than the left eye. Once after the use of a warm blower to dry the hair the conjunctiva became injected. The corneal lesion developed after exposure to extreme cold. As it failed to heal promptly, the lids were sutured together; keratitis subsided, leaving

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Read at the thirtieth meeting of the Society of Neurological Surgeons, Montreal, Canada, May 31, 1937, and at the meeting of the Canadian Medical Association, Ottawa, Canada, June 23, 1937.

1. Verhoeff, F. H.: The Cause of Keratitis After Gasserian Ganglion Operations, Am. J. Ophth. **8**:273, 1925.

2. Buller, F.: A Protective Bandage for the Eye, Lancet **1**:690, 1874.

extensive corneal scarring. When she returned to our care the lids had been closed for seven months.

Direct observation verified her impression that the eye was dry, but it was obvious that some secretion of tears was present. The adhesion of the lids was divided so that more complete examination could be carried out. There was extensive central scarring of the cornea in the anterior layers; the conjunctival vessels were injected, especially on the nasal side, and vision was 6/30 with correction. Measurements proved that secretion of tears was present on the involved side, but it was only one sixth of the amount in the left eye.

Both canaliculi of the right eye were closed by electrocoagulation. They remained blocked for two days, during which time the eye was wet. On the third day the eye appeared dry again, and the canaliculi were proved to be open by syringing. With the drying that followed, pitting of the epithelium was first noticed. The lower canaliculus was then slit by the actual cautery, and it became permanently closed. The eye was more moist but not as much so as it had been immediately following electrocoagulation of both canaliculi. Therefore, the upper canaliculus was subsequently treated with the actual cautery. Though this did not close the canaliculus permanently, it produced ectropion of the punctum which effectively blocked drainage. Since then the eye has been wet but not too troublesome, and the pitting of the epithelium has disappeared, as has the congestion of the conjunctival vessels. The subjective sensation of discomfort, which was present even when the lids were sutured and during the time when the eye was dry, is no longer noticed. The patient was aware of an improvement in vision, which is now equal to 6/15 with correction.

COMMENT

The satisfactory result following closure of the canaliculi has been maintained over a period of five months. It is comparable to the results described by Beetham [3] after he had closed the ducts in patients with filamentary keratitis.

Although closing the canaliculi would seem to be a simple procedure, owing to the fact that sensation is lost and no anesthetic is required, the canals tend to become patent again. When the canaliculus was slit with the actual cautery it closed permanently, and therefore such a procedure would seem to be the one of choice.

The incidence of neuroparalytic keratitis after complete section of the posterior root of the fifth cranial nerve or destruction of the ganglion by alcohol is difficult to determine, as it may occur many years after the therapeutic surgical intervention. Wilfred Harris [4] expressed the belief that it probably occurs in about 20 per cent of the cases. Other authors have stated the belief that 10 per cent is a fair estimate, but both these percentages seem high to us.

If the important factor in the development of the corneal lesions is diminished secretion, it should be possible by detailed physiologic tests to select the cases in which this complication is likely to develop and to prevent it.

3. Beetham, W. P.: Filamentary Keratitis, Tr. Am. Ophth. Soc. **33**:413, 1935.
4. Harris, Wilfred: The Facial Neuralgias, London, Oxford University Press, 1937, p. 62.

We have studied the lacrimal and parotid secretion in every patient who has been operated on for trigeminal neuralgia in the Montreal Neurological Institute in the past year. Measurement of the secretion of tears has been carried out by placing twisted absorbent cotton pledgets in the internal canthus of each eye so that they cover both puncta. These pledgets are allowed to remain in situ for five minutes. The difference in weight in milligrams before and after insertion is taken as an index of the amount of secretion. To simplify the procedure and make it as accurate as possible the dry cotton pledgets are placed in glass weighing jars with ground glass stoppers and weighed. The pledgets are then removed with a forceps from the jars and placed in the eyes, then returned to their respective jars and covered with the ground glass stoppers to prevent evaporation and weighed immediately. We have used the Sartorius analysis suppression scales, which weigh to 0.1 mg., and they have always been operated by the same technician. For the measurement of parotid secretion the same technic has been used, except that nasal tampons have been placed over Stensen's ducts, as suggested by Poth.[5]

When repeated tests have been carried out on a patient the results, as might be expected, have varied. We have obtained, however, definite information as to the presence of secretion and a fairly accurate impression of its amount. We have felt that the weighing method when done properly is more valuable than Schirmer's[6] test with filter paper. It has been surprising to find that the secretion from the parotid gland is usually reduced on the affected side when the lacrimal secretion shows reduction. Since this measurement is a coarser procedure and causes less discomfort to the patient it may eventually prove to be the more valuable test, but more data are required before this conclusion can be drawn.

The problem of the secretory nerve supply to the lacrimal gland need not be considered here. It is as complex as that of the secretory nerve supply to the parotid gland, and our studies on patients have not served to clarify it. For example, Verhoeff's hypothesis that diminished secretion of tears is due to injury of the greater superficial petrosal nerve is contradicted by the findings in the case we have reported. In this case neither the greater superficial petrosal nerve nor the facial nerve was injured; yet there was diminished lacrimation, and keratitis developed. If the sensory loss were the all-important cause, the lesions should appear as a complication much more frequently than they do. We can see no reason for accepting a so-called trophic influence, as

5. Poth, E. J.: A Simplified Technique for Quantitative Collection of Salivary Secretions of Man, Proc. Soc. Exper. Biol. & Med. **30**:977, 1933.

6. Schirmer, O.: Studien zur Physiologie und Pathologie der Tränenabsonderung und Tränenabfuhr, Arch. f. Ophth. **56**:197, 1903.

first suggested by Magendie [7] and more recently advanced by Tagawa.[8] This complex theory has no adequate proof, in our opinion.

Another example of keratitis due to drying is keratitis e lagophthalmo. It is seen commonly in the comatose patient who lies with the eyes open or only partially closed, with no blinking and no conscious discomfort. Corneal lesions seldom develop after paralysis of the seventh nerve. Although the patient does not wink, the discomfort which drying causes stimulates him to roll the eye up under the lid and thus moisten the cornea. The drying of the cornea occurs chiefly during sleep, but even then the eye tends to roll up so that the cornea is partially covered. The lack of winking and ectropion of the lower punctum present after palsies of the seventh nerve, with the consequent damming back of tears, must also be a beneficial factor.

Years ago Magendie observed in animals that the eye in which keratitis developed following section of the fifth nerve was dry. He dismissed drying as the cause of the lesions, because when he took out the lacrimal gland in other animals corneal lesions did not develop. It is now recognized that corneal lesions do not develop after excision of the lacrimal gland because the secretion of the accessory lacrimal glands, such as Krause's glands, is usually adequate. In keratomalacia, though the eye appears to be dry this is not really the case.

The occurrence of keratitis after total rhizotomy for the relief of trigeminal neuralgia is such a serious complication that the following routine is suggested: Oil should be dropped in the eye immediately after operation and a Buller's shield applied. The shield should remain in place until it can be determined that the eye is not dry. If the secretion of tears is adequate to prevent injection of the conjunctival vessels when the eye is left uncovered and every precaution is taken to avoid trauma, no complications are to be anticipated. The patient is discharged from the hospital with Adson and Schroeder's [9] protective shield attached to spectacles. On the other hand, if tears are greatly diminished and the conjunctiva becomes injected, the canaliculi should be closed. This procedure causes no deformity and no special inconvenience. The conjunctival injection should be considered as an elective indication. Corneal pitting or loss of epithelium makes closure of the canaliculi an operation of necessity. Our experience with one case would suggest this to be adequate. Should it fail, tarsorrhaphy is imperative.

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7. Magendie, M.: De l'influence de la cinquième paire de nerfs sur la nutrition et les fonctions de l'oeil, J. de physiol. expér. **4**:176, 1824.

8. Tagawa, S.: Ueber die Beschaffenheit der Hornhautoberfläche nach Trigeminusdurchschneidung, Arch. f. Augenh. **102**:231, 1929.

9. Adson, A. W., and Schroeder, A. A.: A Corneal Shield, J. A. M. A. **86**: 1835 (June 12) 1926.

ORBITAL CYST WITHOUT EPITHELIAL LINING

REPORT OF TWO CASES OF BLOOD CYST

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Cysts without lining of epithelium can be found outside the orbital tissues. Of these, perhaps the best known are hydatid cysts of the liver, blood cysts of the breast, subdural blood tumors of the cranial cavity and, within the eyeball, cysts of the retina and serous cysts of the iris. A few types of cyst without epithelial lining within the orbit and outside the globe have been studied, and several reports of such cysts have been published. Examples of these are serous or exudation cyst, parasitic cyst, congenital cyst of the orbit associated with microphthalmos or with coloboma, cyst in the sheath of the optic nerve [1] and blood cyst. A serous cyst of the orbit may have its origin in the bursa between the tendon of the superior oblique muscle and the trochlea or in the bursa between the levator muscle of the upper eyelid and the superior rectus muscle. Cyst caused by parasites in the orbit must be rare in this country, although a number have been reported in Europe and Asia. A number of cases of congenital cyst associated with microphthalmos and anophthalmos have been reported. A congenital defect in the wall of the eyeball makes the development possible, and, starting in the lower part of the globe, the cyst may grow until it occupies a large part of the orbital cavity. A good example of cyst associated with coloboma is that reported by Calhoun.[2] Several cases of blood cyst in patients operated on for unilateral exophthalmos have been described. I should like to report two cases of this type of cyst.

REPORT OF CASES

CASE 1.—F. R., a 29 year old man, was referred to me by Dr. A. C. Snell because of unilateral exophthalmos. I first saw him on Oct. 11, 1934. About two years before this the right eye had started to be prominent. He gave no history of trauma or inflammation. The bulging of the eyeball had gradually progressed.

From the Institute of Ophthalmology of the Presbyterian Hospital in the city of New York.

Read at the meeting of the Section of Ophthalmology of the New York Academy of Medicine, March 15, 1937.

1. Bane, W. C.: Cyst of Dural Sheath of Optic Nerve, Am. J. Ophth. **1**:17, 1918.

2. Calhoun, F. P.: Bilateral Coloboma of the Optic Nerve, Associated with Holes in the Disk and a Cyst of the Optic Sheath, Arch. Ophth. **3**:71 (Jan.) 1930.

He had been under treatment for inflammation of the right frontal sinus and the antrum. Vision was normal in each eye. The right eye was displaced forward, downward and slightly outward. There was an exophthalmos of about 8 mm. The protruding eyeball showed no pulsation, and auscultation of the skull gave negative results. The patient said that the right eye was irritated and he slept with it half open, that there was watering of this eye, and that he had "pressure headaches." A plotting for diplopia was made, and it was found that the right eye had definite limitation of motility except in turning of the eyes to the right. The basal metabolic rate was normal. The interior of each eye was without abnormality. Roentgen examination by Dr. Pfeiffer showed well defined asymmetry of the orbits, with a pathologic state of the right orbit. The report was as follows:

"In the roof of the right orbit a spherical cavity measuring approximately 35 mm. lies adjacent to the large frontal sinus. The walls of this cavity are thin and are complete on all sides save below, where the cavity encroaches on the orbit. The content of the cavity is of diminished density and impresses me as being nearly clear. The roof of the cavity bulges upward 5 or 6 mm. The right optic canal is circular in contour and measures 5 mm. in diameter. The left optic canal is similar. The ethmoid cells adjacent to the right canal are clouded. The large cavity in the roof of the right orbit is typically that of a retention cyst."

With the patient under anesthesia induced by tribromethanol in amylene hydrate and ether I operated on October 13. Two intermarginal sutures were introduced to hold the margins of the lid in apposition during and for a few days following operation. An incision was made through the skin just below the eyebrow, and a large fluctuant mass was felt in the upper portion of the orbit just behind the orbital margin. The anterior part of the sac was exposed, and an incision was made in it. A large quantity of greenish fluid about the consistency of pus was evacuated. A part of the wall of the cyst was removed for microscopic study. After the fluid contents of the cyst were removed, a thorough application of pure phenol was made to the interior of the wall of the sac, and it was well neutralized by alcohol. The deep structures were brought together by chromic gut sutures, and the incision in the skin was closed with interrupted silk sutures. A pressure dressing was applied in order to hold the collapsed walls of the cyst in apposition, and the pressure was maintained for a week. Recovery was uninterrupted, but there was numbness up to the hair line on the right side of the forehead.

The pertinent part of Dr. Reese's report on the microscopic examination is as follows:

"The specimen consists of dense fibrous tissue, the older being external and that of more recent production mostly internal, with gradations between. Throughout the inner half of this fibrous tissue are many lymphocytes, in some places forming focal accumulations. The internal surface shows no epithelial lining, but instead there are some fragments of fibrin. Over a large portion of this wall plaques of bone have been laid down. There is no evidence of tuberculosis. There is no evidence of malignancy. The impression is that this could well be the result of the organization of a large hemorrhage."

Samples from the contents of the cyst showed no bacteria with either gram stains or acid-fast stains.

This is almost surely an example of blood cyst of the orbit, although there was no history of traumatism. Probably there had been no recent hemorrhage, as the fluid was green rather than red.

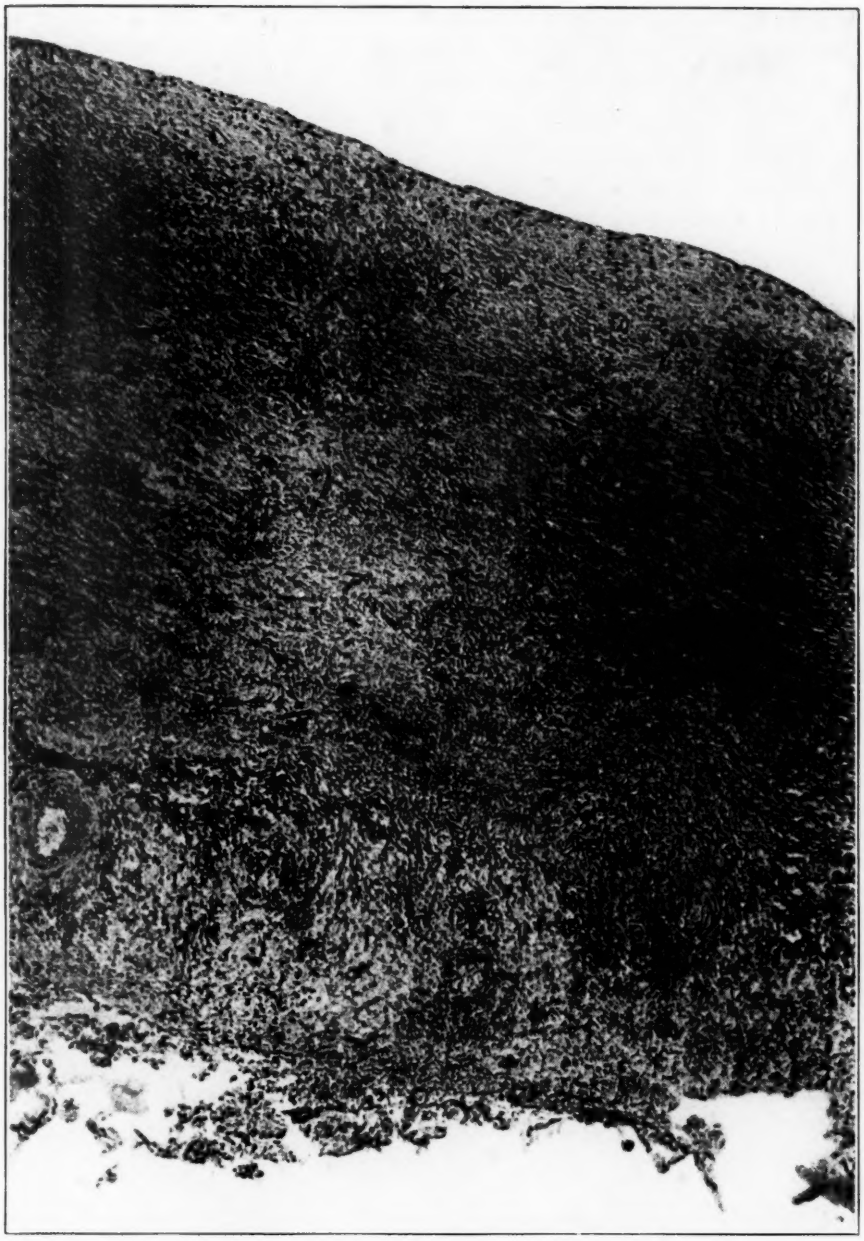
CASE 2.—On Dec. 24, 1936, Dr. Charles A. Elsberg referred to me J. H., a man 45 years of age. In April 1935 during a routine physical examination Dr.

Theodore Sanders noticed bulging of the right eye. The patient was referred to an ophthalmologist, who thought the patient should be in the hands of a surgeon specializing in work on the brain, and he was sent to Dr. Elsberg, who kept him under observation until he was referred to me. During the early months of proptosis a tentative diagnosis of lipoma of the orbit had been made. Roentgen therapy had been administered, without benefit. A few days before I saw the patient, while playing with his son he had experienced the feeling that the right eye was "popping out" and the upper lid caught back of the eyeball. On December 24 vision of the right eye was 20/20 — 4 and that of the left eye 20/15 — 2. The patient accepted a + 1.00 D. sph. ⊃ + 0.25 D. cyl., ax. 55 for the right eye, with which vision was 20/20, and a + 0.25 D. sph. for the left eye, with which vision was still 20/15 — 2. There was exotropia of 20 degrees for distance and 25 degrees for near vision. A plotting for diplopia showed exotropia in all directions of gaze. The separation of the images was greatest when the eyes turned to the left and upward, but the imbalance was not far from comitant. There was exophthalmos of the right eye of 6 mm. The palpebral fissure was longer and wider in the right eye than in the left. The measurements of the width of the fissures were as follows: with the eyes looking straight ahead, 14 mm. for the right eye and 9.5 for the left eye; with the eyes looking up, 17 mm. for the right eye and 11.5 mm. for the left eye, and with the eyes looking down, 6 mm. for the right eye and 4.5 mm. for the left eye. The lower lid did not adhere closely to the globe for about 7 mm. from the outer canthus. The conjunctival vessels appeared dilated behind the equator, but there was no discoloration of the conjunctiva. Between the globe and the lateral orbital wall was a smooth, nonlobulated round mass which protruded slightly beyond the orbital rim; this protrusion held the outer part of the lower lid slightly away from the eyeball. Protrusion of the mass was increased by pressing the eyeball back into the orbit. There were no pulsation and no subjective or objective bruit. Fluctuation was easily elicited. The blindspots and the outlines of the fields were normal. The interior of each eye was normal. Dr. Dyke as a result of roentgen examination at the Neurological Institute made a tentative diagnosis of cyst of the right lacrimal gland. My diagnosis was orbital cyst below the lacrimal gland.

Dr. Pfeiffer gave the following report on the results of roentgen examination:

"Films of the orbits show the margins to be asymmetrical, the left to be normal and the right to be larger and to have a defect in the frontal process of the zygoma. This defect is concave, measures approximately 11 mm. and extends into the bone for 4 or 5 mm. and, seemingly, posteriorly for a short distance. The temporal line is normal. The margins of this defect, or area of diminished density, are irregular and may not be palpable. The right orbit seems slightly larger and shows increased density of soft tissue. The optic canals appear to be circular and symmetrical and measure approximately 5 mm. in diameter. All the paranasal sinuses are large and clear. The sella turcica measures 9 by 12 mm. and is not eroded."

On December 29 I operated on this patient while he was under anesthesia induced by tribromethanol in amylene hydrate and ether. A horizontal incision was made from the outer canthus backward over the zygomatic bone. The tarsoorbital fascia was freely incised along the orbital margin, both above and below the horizontal incision, and the conjunctiva was separated from the globe on the temporal side. A smooth-surfaced, brownish, fluctuant mass could be seen and felt. It was freed on the medial surface by blunt dissection with the finger and the curved scissors. It was not attached to the lateral rectus muscle but to the outer wall of the orbit from the orbital margin back about 30 mm. It rested on the floor



Photomicrograph of the wall of the orbital cyst in case 2. There is absolutely no lining of epithelium or endothelium.

of the orbit but was not adherent to it. By mistake the wall of the cyst was ruptured, and a quantity of red fluid escaped. The fluid was only a little darker than fresh arterial blood. The unattached portion of the wall of the cyst was dissected out and sent to the laboratory for examination. Palpation of the lateral wall of the orbit revealed a depression approximately 6 mm. in diameter, with a rim of firm tissue along the upper border of the depression. The part of the cyst attached to the outer wall of the orbit was sponged dry and treated with pure phenol; this was followed by washing with alcohol. The septum orbitale was closed with catgut sutures, and the incision in the skin was closed with fine silk sutures. Before the dissection was started, two silk sutures had been placed to hold the margins of the lids together. These were allowed to remain, and a pressure dressing was applied. Healing was uninterrupted.

Dr. Reese gave the following report on the microscopic examination of the wall of the cyst:

"The specimen is the wall of a cyst in which *no epithelial elements are noted* (the appearance is shown in the figure). The tissue is merely a fibrous wall, the inner layers of which are edematous. Throughout all the layers there is an infiltration of lymphocytes and a few polymorphonuclear leukocytes. Particularly noticeable is a broad and definite layer of pigment-bearing cells which occurs just beneath the internal surface of the wall. The pigment has the appearance of being of a hematogenous nature and has resulted from the phagocytosis of hemosiderin crystals from old blood. There are some cholesterol crystals in the wall of the cyst, and a few are surrounded by foreign body giant cells. There are also pus cells, lymphocytes, various larger unrecognized cells which are probably edematous fibroblasts, and other types of cells. There is no caseation. Sections stained by Perl's method show hematogenous pigment, and those stained by the Fontana method show no melanin."

Dr. Purdy Stout studied the slides and reported as follows:

"The lining of the cyst is made up of fibrous tissue in which there are many phagocytes full of blood pigment, foam cells loaded with lipoid, and many capillaries in isolated areas. There seems to be some degeneration but scarcely any evidence of inflammation. This is the picture which one sees in cyst that develops from areas of traumatic necrosis of fat. My associates and I have seen this type of cyst in the breast in persons from whom no history of trauma could be obtained. I cannot offer any other explanation for this cyst."

Dr. Karl Meyer examined the fluid content of the cyst and reported that it was of red blood color and contained innumerable intact red blood cells. There were also abundant typical plates of cholesterol. On dilution with saline solution a thick film of cholesterol crystals floated on the surface and adhered to the sides of the container. The quantity of cholesterol was found to be 15 per cent of the total dry weight.

On March 9, 1937, a little over two months after operation, the exophthalmometer readings were 22 mm. for each eye. The diplopia had changed little.

COMMENT

In case 2 probably there had been a fresh hemorrhage into the cyst a few days before I saw the patient, when the eye had a "popping out" feeling, and the fresh hemorrhage accounted for the blood red color of the fluid contents. In case 1 probably there had been no bleeding into the cyst for a relatively long time before operation, and so the contents were not red but green.

In neither of these cases was a significant history of traumatism elicited, and it may be that traumatism is not necessary for the development of a blood cyst within the orbit. A blood tumor in the breast may appear in an area involved in traumatic necrosis of fat, or it may appear in a patient without a history of trauma. A persistent subdural hematoma may occur without direct traumatism to the area involved.[3]

In 1932 Crigler [4] reported a subchoroidal hemorrhage in the left eye of a woman 44 years of age who fell to the floor, landing on her right knee, with only an indirect jar to the head.

Against this there may be a definite history of orbital injury, as in the case of Denig's [5] patient, who received an orbital injury while fighting a duel.

Awerbach [6] reported three cases of hemorrhagic tumor of the orbit, in two of which there was a definite history of injury years before he examined the patients.

Michaïl [7] reported a blood cyst that resulted from the presence of a wad of cotton sponge that was left in an orbit at the time of an enucleation. He operated on the blood cyst ten years after the enucleation.

Vasomotor disturbances (such as those occurring in association with the menopause), hemophilia and alteration in the endothelium of the orbital blood vessels have been mentioned in connection with the etiology of hemorrhage into the orbit.

In a discussion of blood cyst of the orbit attention might be called to the fact that there may be bleeding into an epithelium-lined cyst, giving the contents a red color which is evident when the wall of the cyst is opened. Authors have called attention to defects in the epithelial lining of walls of a dermoid cyst. At the meeting of the American Ophthalmological Society in 1936 Bernard Samuels [8] reported that among thirteen dermoid cysts he found defects in the epithelium in the walls of three. The epithelial lining was broken by patches of granulation tissue, and it is likely that hemorrhage may occur in such areas from newformed vessels.

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3. King, Clarence: Chronic Traumatic Subdural Hematoma as a Cause of Choked Disc, Am. J. Ophth. **20**:149, 1937.

4. Crigler, L. W.: Subchoroidal Hemorrhage Diagnosed as Sarcoma of the Choroid, Arch. Ophth. **8**:690 (Nov.) 1932.

5. Denig, Rudolph: Subperiostal Bloodcyst of the Orbit: Report of a Case, Ophth. Rec. **11**:187, 1902.

6. Awerbach, M. I.: Les tumeurs sanguines de la cavité orbitaire, Ann. d'ocul. **170**:863, 1933.

7. Michaïl, D.: Cyste hématique de l'orbite, Arch. d'opht. **52**:851, 1935.

8. Samuels, Bernard: Dermoid Cysts of the Orbit, Arch. Ophth. **16**:776 (Nov.) 1936.

In most cases of hemorrhage into the orbit absorption of blood occurs, and it is not known why occasionally a wall of connective tissue forms around the blood. Neither is it known why blood within the sac may be found unclotted.

Osmosis may be a factor in the growth of a blood cyst in the orbit, but probably in most cases growth comes through recurrent hemorrhages. Probably the wall is formed as a result of reaction of the orbital tissue to the presence of material foreign to it.

Removal of the entire wall of the cyst is not necessary, whether there is any epithelial lining or not. Generous application of phenol will destroy epithelium, endothelium or the surface of connective tissue. Then if heavy pressure is applied over the lids (while they are held together by intermarginal sutures) there will be apposition of the walls of the cavity and healing between them.

In operating the surgeon can get a good exposure of the orbital contents without an osteoplastic flap, as in the Krönlein procedure. Free canthotomy, with continuation of a horizontal incision beyond the orbital margin and with free incisions of the tarso-orbital fascia along the orbital margin above and below, will allow of good retraction of flaps, with exposure of the wall of the cyst.

DISCUSSION

Dr. Algernon B. Reese: Dr. Wheeler has described an unusual type of orbital cyst. In passing he has mentioned another rare type of cyst of the orbit which also has no epithelial lining, that is, the congenital cyst associated with coloboma, or retinocele. This usually is found in association with microphthalmos, and then the diagnosis is obvious. Occasionally it is present in an eye of normal size and appearance, and then the diagnosis may be difficult. Two microscopic specimens of such a cyst were shown. The cyst may be larger than the eyeball. The wall is composed of tissue resembling that of the sheaths of the optic nerve and is lined by neuroglial tissue and sometimes by retinal elements. The cyst produces exophthalmos which is noticed usually at birth but which increases after birth so that it may lead to an ulcer e lagophthalmos. Determination of the condition of the optic nerve usually leads to the correct diagnosis. The nerve head may show the congenital remains of tissue over its surface, or there may be a colobomatous cupping. The question of an orbital growth or inflammation as a possible etiology may arise.

LATE RESULTS OF EXTRACTION OF CATARACT

EDWARD JACKSON, M.D.
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The late results of extraction of cataract cannot be known from the statistics of great operative clinics, whether situated in India, Vienna, London or America. To such clinics patients go from long distances, to remain until the operative wound may heal and sometimes until a first trial of vision with glasses can be recorded. Rarely does the patient return to such a clinic to report the ultimate, or late, results of his treatment. The permanence of the resulting vision and its total value to the patient can be more certainly judged by the results in patients seen in private practice and followed to the end of life or at least for some years after the operation.

REVIEW OF CASES

A diabetic patient with double cataract went to Vienna, where one lens was extracted. The next year he went to Wiesbaden, where the other lens was extracted. He came to me eighteen months later to see if his vision could still be improved. He showed successful operative results in each eye. The extractions had been done with iridectomy, the capsule being left. He wore glasses that had been prescribed a few weeks after the last operation—spherical lenses, giving vision of 0.3 in each eye. Lenses were prescribed fully correcting the astigmatism. These gave vision of 1.3 in each eye, and this vision continued until his death, three years after his last operation.

In the other patient with diabetes included in this series the condition was mild, and there were no retinal lesions or much general disturbance. The patient lived three years after the extraction; vision decreased gradually from 0.5 to 0.25, and she gave up reading. Years ago a patient was seen in consultation who died in diabetic coma a week after extraction of cataract. The condition was severe, but the patient wished to see again before she died. That was before insulin was available to control the condition for operation and even before dietetic treatment had been brought to its present efficiency.

A woman of 65 had been blind in the right eye from cataract. She had it operated on, but an opaque capsule, not effectively divided, left the vision still poor. In another city she had extraction of cataract done on the left eye, and one month later had vision of 20/100 in this eye. But vision in this eye progressively failed. Five months later she came to me, with vision of perception of movements of the hand and chronic uveitis that had left vitreous opacities. Five years later her right eye has vision of 0.25 with her correcting glass. But the left eye is hopelessly damaged, probably from use too soon after operation. There was no other evident cause for the uveitis.

Neither of these extractions was done by me. But the results were evident, and such late results must be considered in forming a just

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estimate of the value of extraction of senile cataract. When living in Philadelphia I observed cases of slow failure of vision after extraction of cataract done by surgeons of the highest skill. A few such failures are due to atrophy of the optic nerve or disease of the central nervous system. But the results in the majority of cases impressed the lesson that ample time, often several months, must be allowed the eye for recovery after operative treatment that so disturbs its whole nutritive mechanism. In a few cases in which both eyes had been subjected to extraction of cataract it was the eye that required the longer time for recovery that showed the better result.

*Table of Cases**

Case	Age of Patient, Years: At Operation	Age of Patient, Years: When Seen	Complications	Vision: Before Extraction	Vision: After Extraction
1	53	68	Myopia	Perception of light	0.80
2	37	75	Myopia; uveitis; corneal scar	Perception of light	0.50
3	61	63		Perception of light	1.10
4	48	49	(Traumatic cataract)	Perception of light	0.40
5	65	72	Uveitis	Perception of light	0.25
6	66	69	Myopia	Perception of light	0.90
7	75	77	Myopia	Perception of light	0.80
8	42	48	Diabetes	Perception of light	1.20
9	68	70	Asthma	Perception of light	0.70
10	60	62		0.03	0.80
11	52	65		0.10	1.00
12	63	75		Perception of light	0.60
13	66	68	Glaucoma	Perception of light	0.80
14	54	59	Corneal scar	0.06	0.50
15	32	34	(Traumatic cataract)	Perception of light	0.30
16	36	41	Tetany	0.03	1.00
				0.20	1.10
17	57	71		0.04	0.20
18	54	70	(Traumatic cataract)	Perception of light	1.20
				Perception of light	1.20
19	32	43	Tetany	0.20	1.10
				0.30	1.10
20	64	67		Perception of light	1.00
21	35	62	Uveitis and glaucoma	Perception of light	1.20
22	57	77	Myopia	Perception of light	1.00
				Perception of light	1.20
23	70	91	(Hypermature cataract); glaucoma	Perception of light	0.50
24	72	75	Diabetes	0.10	0.50
25	60	68	Myopia; orbital cancer	0.05	0.80
26	70	72		0.07	0.60
27	77	80		Perception of light	0.45
28	71	73		Perception of light	0.45
29	73	78		Perception of light	0.80
30	74	84		Perception of light	1.00
				Perception of light	0.50
31	65	70		0.10	0.90
				0.10	1.00
32	74	76		Perception of light	0.50
33	64	68	Myopia	Perception of light	0.80
34	66	71		Perception of light	0.80
35	61	72		Perception of light	1.10
36	55	61		0.10	1.10
37	60	62	Myopia	0.15	0.80
38	50	52	Myopia	0.01	0.40
39	60	67	Tobacco amblyopia	Perception of light	1.00
40	78	80	Loop extraction	0.03	0.20
41	68	70	Loop extraction	Perception of light	0.10
42	83	93		Perception of light	0.12
43	72	77		Perception of light	0.60
44	43	48		Perception of light	0.60
45	75	77		Perception of light	1.00
46	70	72		0.10	0.40
47	73	75		Perception of light	0.90
48	56	65	Glaucoma	Perception of light	0.10
49	54	64	Myopia; uveitis	Perception of light	0.60

* Resulting vision was 1 or more in nineteen eyes.

Among the cases that furnish the chief basis of this paper were four in which cataract was complicated by glaucoma.

In one of these cases a woman of 56 suffered from facial erysipelas, and while she was recovering from this bilateral acute glaucoma developed. Iridectomy for glaucoma was done on the left eye, about one fifth of the iris being removed. The right eye seemed to recover under the use of physostigmine. About a year later the patient was taken to a prominent general surgeon, who had also been one of the surgeons of the Wills Hospital, to see if the right eye should also be operated on. The surgeon wrote to the patient's family physician that there was no sign of glaucoma in either eye and, except for the iridectomy wound, there was no sign that she ever had glaucoma, but that she was getting cataract. Cortical striae were noted in both lenses at the time of the iridectomy. Ten years after that I was called to see her for the cataracts. There was absolute glaucoma in the right eye, but the left eye had good perception of light. Extraction of a mature gray cataract gave her vision of 0.1, which she had retained two years afterward.

Of the other patients with glaucoma, one had glaucoma before the extraction, and the condition was probably chronic; vision was little improved by operation. Two patients had moderately increased tension following needling operations for capsular opacity, which quickly subsided under the influence of miotics. One patient had a sharp rise of tension after preliminary needling of the lens. Extraction was done a few hours later. The patient has had no evidence of glaucomatous attacks in the twenty-seven years that have followed.

For the four cataracts associated with tetany, treatment with calcium and parathyroid preparations and thyroid extract was tried for some months, without any decidedly beneficial effect. Extraction of the cataracts brought restoration of standard vision in all four eyes, which continued for the two to five years that the patients remained under observation.

Myopia has been included among the causes of cataract. The findings in these cases give some support to that idea. But the proportion of previously myopic eyes in the patients, eleven in fifty-five, is not much greater than the norm of 18 per cent for all eyes after the age of 50 years.[1] The late results in these cases seem to indicate that such eyes bear extraction of cataract as well as most other eyes. In case 49 both eyes had been affected by chronic uveitis, and the fellow eye had become entirely blind, and had been enucleated. But since extraction of the cataract vision in the eye has improved until now it is 0.6, and the patient can do her own reading, which was previously impossible.

In case 2 there had long been uveitis with myopia. The eye on which extraction was done had been the worse eye and had not been used for reading, until the other eye had the macula damaged by a choroidal lesion. Extraction of the lens has given it vision of 0.5, and the patient uses her eyes more freely than she has for many years.

1. Jackson, E.: Norms of Refraction, Tr. Sect. Ophth., A. M. A., 1931, p. 179.

Eyes blind from uveitis often show cataract eventually. In case 4 one eye had been lost in this way after the extraction. But the other eye has vision of 0.3, and is still improving after seven years. The important point is that most causes of uveitis can be removed entirely, and if this is done long enough before the operation the history of previous uveitis is no bar to the extraction of cataract. The patient in case 21 had uveitis, for which he was treated at the Hospital of the University of Pennsylvania, ten years before he had cataract. There was no recurrence of the uveitis at any time. Twenty-seven years after the removal of the cataract vision in that eye is as good as that in the fellow eye, which has never been diseased. Furthermore, with the full correction for each eye, differing about 9 D. for the two, the patient has little difficulty when he uses both eyes together.

It should be noted that in all the cases the operation was done with opening of the capsule in my usual manner, and in only a few was there need of later division of the capsule. It was interesting to note in Colonel Wright's lectures that at Madras, India, where the result had to be judged shortly after extraction, capsulotomy was judged the safer operation. In no case in this series was there any detachment of the retina or of the choroid. I have seen these complications in a few cases of extraction of cataract done by other surgeons, but they have never followed my extractions of cataract, except in two cases, in which the eye was destroyed by expulsive hemorrhage. In three cases of this series the operation was done with the patient sitting up in a high-backed chair, in which he was kept for many hours, to prevent expulsive hemorrhage, which there was reason to fear. Twice I have seen such hemorrhage checked by raising the body to the erect position and letting the feet drop at the side of the operating table.

TECHNIC OF OPERATION

The methods and technic of operation have been held to determine the results of extraction of cataract. This is largely true for the immediate, or early, results, and they may also be responsible for the late results. They may properly be considered with reference to this series of cases. In case 2 the operation was done by Dr. Allen Greenwood; in cases 19 and 49 both extractions were done by Dr. Wilmer. In the other cases extraction was done by me, and certain features of the operations may be properly considered to have influenced the results.

The cornea has more rigidity than the sclera. The cornea deserves a name that allies its physical properties with those of horn. The sclera is simply a very thick, tough but flexible membrane. When the corneal incision is made to extend at both ends into the sclera the advantage of the corneal rigidity is lost, and when pressure is applied to the eye the

corneal flap slides up on the part attached to the sclera. If the incision is made at the limbus, with a Graefe knife, it tends in the middle, which is cut last, to drag the sclera out of shape and thus disturb the relations of its contents, the ciliary body, choroid, retina and vitreous. And when the diaphragm between the aqueous and the vitreous is destroyed by intracapsular extraction the effect of this disturbance is increased and may be made permanent. This probably is the cause of the hammock, or updrawn, pupil.

Recognizing that the Graefe knife is excellent for fixing the puncture and counterpuncture but poor for completing the incision, I early designed a modified knife for extraction of cataract. This was first used on the patient in case 48 in 1887, and its improved form was described in the Transactions of the American Ophthalmological Society in 1890.[2] This knife was used in all the cases of this series except three. The incision was made with the puncture and the counterpuncture exactly in the limbus. Then, when it seemed desirable to make a conjunctival flap, the edge was turned a little toward the sclera as the incision was completed, and brought out beneath the conjunctiva. This produced a rather narrow flap but one that seemed to serve as efficiently as a broader flap.

The opening of the capsule of the lens was done with the cataract knife, not while making the corneal incision, but afterward. The knife was dipped in boiling water to cleanse it, and the back was introduced into the corneal incision and drawn back until the point was at the temporal edge of the pupil. Then the point was made to penetrate the capsule of the lens and pushed forward, making an incision in the capsule parallel to the corneal incision. This incision always permitted easy expulsion of the lens, and this procedure was rarely followed by need of a secondary operation.

Corneal or conjunctival sutures were not used for closure of the wound; nor was a tendon suture used for fixation. Fixation was made complete by grasping the conjunctiva with the fixation forceps just below the point for the counterpuncture. The impression grew on me that in doing this operation the anterior capsule always retracted and never gave trouble. Only when there was hemorrhage into the anterior chamber or an inflammatory exudate was subsequent needling required.

The nutrition of the eye is in several respects highly specialized and should be disturbed as little as possible by any operative procedure. Constant respect for the limitations this imposes seems to me the most important condition for operations for cataract, if they are to give the best late results.

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2. Jackson, E.: Tr. Am. Ophth. Soc. **9**:145, 1900.

ABSTRACT OF DISCUSSION

DR. ALLEN GREENWOOD, Boston: It is difficult to cover this subject without some reference to the type of operation and the early results. It is apparent that if the eye is healthy prior to an operation for cataract and there are no accidents at the time of operation and no complications during the healing process the late result is almost uniformly good, especially if one limits oneself to consideration of the results present six months or more after the operation. I have a number of patients who were operated on from fifteen to twenty years ago who had uncomplicated cataracts removed without any accident or complications and who still have the vision which they had shortly after the operation. In view of the fact that the majority of operations for cataract are done on patients who have reached and gone beyond their three score years and ten, many of them do not live long enough to have their late results evaluated. The majority of patients who have had uncomplicated operations rarely need to change the final glass given to them.

The ordinary ocular disturbances of elderly people should not be considered as late results of the extraction. Of course, hemorrhages and conditions that accompany diabetes and renal disease, arteriosclerosis and senile choroiditis may occur. Loss of vision produced by such conditions can hardly be listed among the late results of extraction of cataract. Barring such conditions, it has been my experience that after a year has elapsed since the operation, complications such as separated retina, glaucoma and iridocyclitis are rare. When, however, one takes into account patients who do badly at the time of the operation or have disturbing complications immediately after, one may find after the first six months a lessening of vision. Patients with a portion of the iris or bits of capsule adherent to the wound and those who have lost considerable vitreous may at any time have postoperative complications.

After looking over my records I have come to the unexpected conclusion that patients who have had extraction of the lens in the capsule in a healthy eye are in after years somewhat better off than those who have had an extracapsular operation, in spite of the fact that many of these patients have marked iridodonesis. Several times I have operated on a fully mature cataract in one eye by the extracapsular method and shortly after have removed the less mature lens from the other eye by the intracapsular method.

During a period of two and a half years forty-five operations for cataract were performed; the late results of these were good, and the patients had resultant useful vision, which they have retained, except in three cases.

To summarize: Given a healthy eye, a healthy body, no accidents at the time of extraction and no complications during the process of healing, the late results of extraction of cataract should be almost uniformly good, as far as actual results of the operation are concerned.

Given a sick eye, or one previously diseased, a sick body, accidents at the operation or complications following it or during healing, the late results are very uncertain, but even in such cases they may be surprisingly good.

DR. EDWARD C. ELLETT, Memphis, Tenn.: It is a matter of great concern to persons who undergo an operation for cataract as to what their prospects are for retaining the vision which they regain by the operation. I have made a survey of my private patients with cataract whom I was able to examine or have examined after an interval of five years after the operation, and I have reliable data on one hundred and eighty-eight such patients. Only those were considered in whom vision of 6/9 or better was obtained at the time of dismissal. It would not be fair to blame the operation for the failure of the restoration and preservation of vision in an eye already affected with glaucoma, chronic uveitis or high myopia.

Of the patients considered in this survey, 112 were operated on from five to ten years before, 67 from ten to twenty years before and 8 from twenty to thirty years before, and 1 was operated on over thirty years before. The ocular health of these patients was just as good at the end of the period which had elapsed since their eyes were subjected to the operation as it was in a similar number of patients of the same age on whom no operation had been performed.

There seem to be two conditions especially that often develop after a successful operation for cataract, which are capable of impairing the vision, namely, glaucoma and macular degeneration, neither of which seems to have any relation to the operation or the condition for which the operation was performed.

Doubtless the macular disease was already beginning before the operation but was hard to detect when the cataract was present. To those who have had the unfortunate experience of having met with such cases, the following statement of an operator of the widest experience, Colonel Wright, of Madras, India (*Am. J. Ophth.* **20**: 376, 1937) is comforting: "Posterior-segment lesions are sometimes a cause of disappointment, and are not necessarily appreciated by preoperative tests."

Another distressing condition sometimes seen is retinal detachment.

None of these conditions, in my opinion, is in any way dependent on the cataract or its removal, and my observation leads me to think that the chances for an eye from which a cataract has been successfully removed retaining useful vision as long as the patient lives is just as good as for the eye of any person of similar age and a similar state of health.

In this series there were eleven patients who secured an immediate good visual result and subsequently suffered loss or impairment of vision. The causes of this loss or impairment were as follows: glaucoma (all varieties), in 2 cases; retinal detachment, in 2; atrophy of the optic nerve, in 4; retinal hemorrhages and macular degeneration, in 2, and uveitis, in 1.

DR. EDWARD JACKSON, Denver: May I call attention to the fact that this paper has brought out points that may be more important than what was presented in the paper? The summaries of the two discussers would invite all the members to look into this matter and consider the results of our operations. This is a field of investigation to be occupied legitimately and with the prospect of getting information.

I wish to speak of the operation that was practiced in all these cases. It was done with a knife, the point of which is like that of a Graefe knife, just wide enough to almost complete the corneal section. The peculiarity of the knife is that it has a curve. First I used a knife that was about 4 or 4.5 mm. broad at the broadest point, and completed the

corneal section by pushing forward, but I came to the conclusion later that it was better to use a narrower knife and if there was a slight bridge of tissue to divide that as the knife was withdrawn, pulling the point up to cut back. When one penetrates the cornea, to make the puncture or the counterpuncture, the knife, having a long angle of cutting edge, strikes the cornea obliquely, and little force is required. If one completes the incision with the Graefe knife, which most operators do by making a sawing motion, changing direction two or three times, one pulls up the sclera, particularly if one makes an incision that goes out into the sclera. I thought this caused too much disturbance to the eye.

Fixation was made in the direction that the knife was pointing when it made the puncture and the counterpuncture. The tendency of the eye to rotate before the pressure was made was entirely balanced, and very easily balanced, with little force applied at that point.

Then the incision was made in the capsule. After I tried this procedure and became familiar with it, it seemed to me it was superior to anything else, and I continued to use it. The knife is brought into the corneal section, after it has been dipped in boiling water to sterilize it, and is drawn back until the point is at about the temporal edge of the pupil. Then it is pushed forward in the capsule of the lens, dividing the capsule from that point parallel to incision in the cornea.

The smooth, prompt healing of that sort of incision justifies it, and justifies mentioning it now, forty-seven years after the operation was described, although comparatively few have adopted this form of knife.

Certainly the point that the general structure and the relations of the eyeball should be as little disturbed as possible in the operation for cataract is an important one to have in mind in every manipulation of the instruments that one makes at the time.

A RARE COMPLICATION FOLLOWING APPENDECTOMY

REPORT OF A CASE IN A FIFTY-FOUR YEAR OLD MAN

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DRAMMEN, NORWAY

It is probable that small cerebral hemorrhages, causing no symptoms, occur not infrequently during general anesthesia. Thus, patients often state that they have noticed some degree of deafness subsequent to being given a general anesthetic. If these statements are correct it must be supposed that hemorrhage in the inner ear has occurred. Cases of cerebral apoplexy such as the one here reported are rare.

REPORT OF CASE

K. T., a 54 year old carpenter, was brought to the hospital in Drammen on March 21, 1937. The past history contained nothing of significance apart from a chronic ailment of the nose and ear. The patient had undergone an operation for a deviated nasal septum on February 18. At that time his blood pressure had measured 150 systolic and 110 diastolic. During the night prior to his admission to the hospital he had been seized with abdominal pain. The diagnosis of acute appendicitis was confirmed on admission. Operation was performed within a few hours, with the patient under narcosis induced with scopolamine hydrobromide, morphine and ether. The apparatus of Ombrédanne was used, the customary quantity of ether—100 cc.—being poured into the machine. During the operation the patient collapsed and became extremely cyanotic. After some moments recovery occurred, and the operation was continued, with the patient under the influence of an anesthetic administered by use of the open mask. The quantity of ether used in the aforementioned apparatus was not noted.

When the patient awoke it became apparent that he was totally blind. The attending surgeon also noted paresis of the right arm and leg. The next day I examined the patient. He was a heavy plethoric person, and was restless and somewhat disoriented, so that examination was difficult. The grip of the right hand was weaker then that of the left. The other reflexes could not be satisfactorily examined. Ocular examination revealed complete ptosis of the left upper lid, the right being normal. Each eye was immobile, deviating a little toward the right but not drawn entirely to the canthus. Each pupil was round and moderately dilated and did not react at all to light. The media and fundi were normal. Total amaurosis was present.

The patient soon became increasingly irrational. He died during the following night. Permission for postmortem examination was not secured. A Wassermann test of the blood or spinal fluid was not made.

In summary, a stout man with a somewhat increased blood pressure showed symptoms of a cerebral hemorrhage with resulting total blindness, oculomotor paralysis and, possibly, paralysis of the left abducens nerve.

COMMENT

The cerebral hemorrhage must have been situated in the primary subcortical visual centers on the left side. As will be remembered, this lies in the region of the pulvinar of the optic thalamus, the lateral geniculate bodies and the anterior corpora quadrigemina, below and a little lateral to the aqueduct of Sylvius. In addition, the nerve fibers involved in the reactions of the pupil, in accommodation and in convergence, were affected.

As the ptosis appeared only on the left, the left oculomotor nucleus alone could be damaged. As the pupil of the right eye was also dilated and failed to react to light, the nerve fibers connecting with the right third nucleus must have been injured. As there was no demonstrable vertical deviation of the eyes, a lesion of either trochlear nucleus can be eliminated. On the other hand, a lesion involving the abducens nerve cannot be eliminated. On this account one cannot dismiss the possibility that the hemorrhage was basal and meningeal in origin. It is more reasonable, however, to assume a pontile localization because of the conjugate deviation of the eyes.

So much may be deduced from the position of the eyes and the pupils. Unfortunately, it cannot be definitely stated how far forward or backward the hemorrhage extended. It is, however, unthinkable that a hemorrhage extending posteriorly could reach so far as to involve both optic radiations, with complete blindness as a result, at any rate, without causing death sooner than it occurred in this case. It is easier to suppose that the bleeding extended straight ahead toward the chiasm, destroying its function.

I have been unable to find in the ophthalmologic literature at my disposal a decription of a similar complication following narcosis induced with ether.[1] Poulsson emphasized the fact that chloroform causes a considerable decrease in the general vascular tonus. Ether, on the other hand, is followed by only a slight change in the general blood pressure, apparently only the centers controlling the blood pressure of the face and the surface of the brain being involved.

It is known that narcosis induced with the use of Ombrédanne's apparatus causes temporary asphyxia. It was probably during this state that rupture of a vessel occurred at some point of diminished resistance in the pons. Too pronounced vasodilatation must have taken place, as was indicated by the severe cyanosis. The resulting hemorrhage affected both the oculomotor and the visual centers.

The case is from the service of Dr. Knud Nicolaysen, who permitted me to make the report.

1. Search has included Wilbrand and Saenger's "Neurologie des Auges," 1900-1927, Nelson's loose leaf system, many numbers of the *Klinische Monatsblätter,* the *Zentralblatt der Ophthalmologie* and the *Zentralblatt für Chirurgie,* and the tenth edition of Poulsson's "Lehrbuch der Pharmakologie."

BACTERIAL FACTORS IN CHRONIC CATARRHAL CONJUNCTIVITIS

I. RÔLE OF TOXIN-FORMING STAPHYLOCOCCI

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The etiology of chronic catarrhal conjunctivitis is not clearly defined. With no single causative factor in evidence, a variety of agents and conditions have been regarded as inciting or contributing causes. Among the more important of these are the following: (1) bacteria, principally Haemophilus lacunatus (the diplobacillus of Morax and Axenfeld); (2) viruses, such as those causing molluscum contagiosum and the common wart; (3) acne rosacea; (4) allergy; (5) streptotrichal concretions of the canaliculi; (6) trichiasis; (7) minute foreign bodies; (8) irritants, such as smoke; (9) refractive errors; (10) excessive secretion of the meibomian glands; (11) vitamin deficiency; (12) deficient lacrimal secretion, and (13) chronic nasal sinusitis.

It is proposed in this series of studies to consider the bacterial factors in chronic catarrhal conjunctivitis and to endeavor to determine the relationship of each bacterium to the disease. The term chronic catarrhal conjunctivitis will be used to include all forms of chronic conjunctivitis which cannot be placed in any other category.

RELATION OF BACTERIA TO CHRONIC CATARRHAL CONJUNCTIVITIS

The only universally recognized bacterial cause of chronic catarrhal conjunctivitis at present is H. lacunatus (the diplobacillus of Morax and Axenfeld). Its etiologic rôle has been established conclusively by the inoculation experiments on man of Morax,[1] Axenfeld,[2] Hoffmann,[3] Gifford[4] and Erdmann.[5] That the conjunctivitis is due to the liberation

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From the Institute of Ophthalmology, Columbia University.

Read at the meeting of the Section of Ophthalmology of the New York Academy of Medicine, March 15, 1937.

1. Morax, V.: Ann. Inst. Pasteur **10**:337, 1896; Ann. d'ocul. **117**:5 (Jan.) 1897.

2. Axenfeld, T.: Bacteriology of the Eye, translated by Angus MacNab, London, Baillière, Tindall & Cox, 1908, p. 171.

3. Hoffmann: Arch. f. Ophth. **48**:639, 1899; cited by Axenfeld.[2]

4. Gifford, H.: Ann. Ophth. **7**:218 (April) 1898; Ophth. Rec. **14**:511, 1905; cited by Axenfeld.[2]

5. Erdmann: Klin. Monatsbl. f. Augenh. **63**:501, 1905; cited by Axenfeld.[2]

of soluble toxic products of the bacterium is indicated from the experiments of Morax and Elmassian.[6] These investigators induced conjunctivitis by instilling toxins, produced in culture by the diplobacillus, into the conjunctival sacs of animals and human volunteers. That the toxin must also be liberated in the sac in the natural disease may be deduced from the work of Lindner,[7] Howard [8] and others, who have shown that the diplobacilli do not invade the epithelium but proliferate only in the secretion and on desquamated epithelium. The researches of Erdmann [5] and others have shown, further, that diplobacilli may occasionally occur on the healthy conjunctiva. Thus two important principles would seem to have been established: (1) A bacterium multiplying only in the secretion can induce conjunctivitis by the liberation of toxin and (2) a known conjunctivitis-producing bacterium may occur occasionally on the healthy conjunctiva.

The occurrence of many other varieties of bacteria has been noted in chronic catarrhal conjunctivitis. Some of these, such as Corynebacterium xerosis and other diphtheroids, have been generally considered to be without pathogenic significance. To such well known pathogens as Streptococcus haemolyticus and Haemophilus influenzae, on the other hand, an etiologic rôle has frequently been ascribed, although there have been no substantiating results in inoculation experiments on man.

THE BACTERIAL FLORA IN CHRONIC CATARRHAL CONJUNCTIVITIS

A bacteriologic analysis [9] was made in 399 cases of chronic catarrhal conjunctivitis observed in the ophthalmic clinic of the University Hospitals, Iowa City, and of 244 cases observed at the Vanderbilt Clinic, New York. Only those bacteria considered to be of possible etiologic importance are recorded.

In the New York series the relatively small number of cases in which nonpathogenic bacilli were observed was probably due to the repeated cultures which were made in many instances and to the inclusion of white staphylococci, when hemolytic, in the list of etiologic possibilities.

Noteworthy was the failure to find any cases of diplobacillary conjunctivitis in the New York series. Three strains of diplobacilli morphologically like the bacillus of Morax and Axenfeld proved on culture to be distinct from it but were not identified.

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6. Morax, V., and Elmassian: Ann. d'ocul. **72**:6, 1899.

7. Lindner, K.: Ztschr. f. Augenh. **42**:11, 1919; Arch. f. Ophth. **55**:726, 1921.

8. Howard, H. J.: Tr. Am. Ophth. Soc. **22**:186, 1924.

9. Cultures made on blood agar by inoculation of material obtained from the lower fornix with a platinum loop were incubated at 37 C. and examined after forty-eight hours. In the New York series sterile swabs moistened in broth containing 1 per cent dextrose were used in taking material.

The staphylococcus is seen to be by far the most common bacterium of possible etiologic consequence observed in chronic conjunctivitis in these series, and its significance is the subject of this first report.

TABLE 1.—*Bacteria Noted in Cases of Chronic Catarrhal Conjunctivitis*

Bacteria	No. of Cases
Iowa Series	
Staphylococcus aureus	175
Diplococcus pneumoniae	22
Haemophilus lacunatus (Morax, Axenfeld)	9
Neisseria catarrhalis	4
Streptococcus haemolyticus	4
Streptococcus viridans	2
Haemophilus influenzae	2
Staphylococcus aureus and Haemophilus lacunatus	3
Staphylococcus aureus and Haemophilus influenzae	1
Staphylococcus aureus and Diplococcus pneumoniae	5
Staphylococcus aureus and Streptococcus viridans	1
Staphylococcus aureus and Streptococcus haemolyticus	2
Staphylococcus aureus, Diplococcus pneumoniae and Neisseria catarrhalis	1
Haemophilus lacunatus and Diplococcus pneumoniae	3
Haemophilus influenzae and Aerobacter aerogenes	1
Unidentified gram-negative bacilli	2
Nonpathogenic bacteria (including diphtheroids, Staphylococcus albus and sarcinae)	162
Total	399
New York Series	
Staphylococcus aureus	80
Staphylococcus albus (hemolytic)	78
Diplococcus pneumoniae	6
Haemophilus influenzae	5
Streptothrix Foersteri	2
Streptococcus haemolyticus	1
Proteus ammoniae	3
Staphylococcus aureus and Diplococcus pneumoniae	1
Staphylococcus aureus and Staphylococcus albus (hemolytic)	9
Staphylococcus aureus and Streptococcus viridans	2
Staphylococcus aureus and Streptococcus haemolyticus	1
Staphylococcus albus (hemolytic) and Diplococcus pneumoniae	1
Staphylococcus albus (hemolytic) and Streptococcus nonhaemolyticus	2
Haemophilus influenzae and Aerobacter aerogenes	1
Pseudomonas aeruginosa, Escherichia coli and Klebsiella pneumoniae	1
Unidentified gram-negative bacilli	3
Nonpathogenic bacteria (including diphtheroids, nonhemolytic Staphylococcus albus and sarcinae)	48
Total	244

ARE STAPHYLOCOCCI PATHOGENIC FOR THE CONJUNCTIVA?

History.—Negative results of inoculation experiments with staphylococci on the human conjunctiva have been reported by Leber,[10] Sattler,[11]

10. Leber: Internat. Cong. Ophth., Heidelberg, 1880; cited by Axenfeld,[2] p. 233.

11. Sattler: Internat. Cong. Ophth., Heidelberg, 1880; cited by Axenfeld,[2] p. 233.

Bach [12] and Hirota.[13] A single inoculation with a positive result has been performed by McKee.[14] Morax and Elmassian [6] have demonstrated conjunctivitis-producing toxin in broth cultures of Staphylococcus aureus isolated from the conjunctiva. When instilled into the eyes of human volunteers and of laboratory animals this toxin produced a definite conjunctival inflammation after an incubation period of a few hours. These results would seem to be of primary importance, but in the absence of confirmatory reports some observers, including Lindner [7] and Howard,[8] stated the opinion that staphylococci appear on the conjunctiva only as harmless saprophytes. Others, such as Burky,[15] have expressed the belief that they are an important cause of conjunctivitis.

Inoculation Experiments.—In view of the difficulty which is encountered in interpreting the results of inoculation experiments on the conjunctiva, it would seem that in the case of staphylococci the following conditions for a significant experiment should be satisfied: (1) A virulent, freshly isolated strain must be employed and (2) the staphylococci must multiply on the conjunctiva. The second condition is of prime importance, since staphylococci are known to multiply in conjunctivitis, and any inoculation experiment in which they are rapidly eliminated from the conjunctiva should be considered of little or no significance. Inoculations in man are far more valuable than inoculations in animals, on account of the resistance of laboratory animals to bacterial conjunctivitis.

In the absence of human volunteers a series of inoculations with freshly isolated toxigenic staphylococci from chronic conjunctivitis were made on rabbits, guinea-pigs and monkeys, in groups of six. These animals proved to be wholly resistant to simple deposition of cultures on the conjunctiva.

In a second series, instillations in monkeys and rabbits, followed by vigorous massage with cotton applicators, produced moderate conjunctivitis that persisted for several days. Control eyes, treated by massage alone, showed only temporary irritation.

A third series of rabbits and monkeys were subjected to chemical irritation by instillations of a 2 per cent solution of silver nitrate. Two hours later one eye of each animal was heavily inoculated with staphylococci. The resultant conjunctivitis was much more marked in the inoculated eyes and persisted for several days after the irritation of the uninoculated eyes had healed. In no instance, however, did chronic conjunctivitis develop.

In a fourth series of rabbits fresh cultures were injected intracorneally. The corneal abscesses or ulcers which resulted were always accompanied by a severe purulent conjunctival reaction.

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12. Bach, L.: Arch. f. Ophth. **40**:3, 1894; cited by Axenfeld,[2] p. 233.

13. Hirota, cited by Axenfeld,[2] p. 233.

14. McKee, S. H.: Bacteriology of the Eye, in Wood, C.: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1913, vol. 2, p. 824.

15. Burky, E. L.: Am. J. Ophth. **19**:841, 1936.

The results in these four series of experiments indicate that under certain circumstances staphylococci may be pathogenic for the conjunctiva of laboratory animals, but no chronic conjunctivitis comparable to that occurring in man could be produced, and it was impossible to establish growth of staphylococci in the conjunctiva; the bacteria were always rapidly eliminated.

Toxin Conjunctivitis.—One hundred and fifty strains of staphylococci from material from eyes with chronic conjunctivitis were tested for conjunctivitis-producing exotoxin. The toxins were prepared according to the method of Leonard and Holm,[16] according to which growth is maintained in semisolid agar in an atmosphere of 80 per cent carbon dioxide and 20 per cent oxygen. They were adjusted to a p_H of from 6.8 to 7 and rendered bacteria-free by filtration through Mandler filters.

The conjunctivitis-producing power was determined by instillation of filtrates into the eyes of adult white rabbits at five minute intervals for one hour. Of the 150 filtrates tested, 57 exhibited definite conjunctivitis-producing power. The test conjunctivitis was characterized by (1) an incubation period varying from one to two hours and (2) a purulent inflammation varying in degree from a mild but definite conjunctivitis to an acute reaction with swelling of the lids and closure of the eye. The conjunctivitis subsided in two or three days, leaving no demonstrable immunity.

Two tests on the human conjunctiva showed it to be apparently more susceptible to the toxin than the conjunctiva of the rabbit, a single drop of active toxin producing acute or subacute conjunctivitis with bulbar hemorrhages.

The following evidence indicated that the test conjunctivitis was a true toxic reaction: (1) The uninoculated medium produced no irritation; (2) the toxin, when adjusted to neutrality, produced no immediate signs of inflammation; (3) there was a definite period of incubation; (4) toxin detoxified by formaldehyde, boiling or prolonged alkalinization did not produce conjunctivitis, and (5) immunization by intracutaneous injections of toxin weekly for ten weeks prevented toxin conjunctivitis in two tests on rabbits. (Immunization was not effected by repeated local instillations of toxin.)

Evidence in Favor of the Existence of Staphylococcic Conjunctivitis.—The evidence from this study which indicates that staphylococci are a factor in the production of conjunctivitis may be summarized as follows: 1. Virulent staphylococci have been observed in material from eyes with conjunctivitis in which no other cause could be demonstrated. Representative strains have proved to be pathogenic for rabbits on

16. Leonard, G. F., and Holm, A.: J. Immunol. **29**:209, 1935.

intracorneal and intravenous injection, and toxins produced by them have shown the hemolytic, dermonecrotic and lethal properties characteristic of toxins of pathogenic staphylococci from other sources. 2. A conjunctivitis-producing factor has been demonstrated in toxins from these bacteria. Fifty-seven of 150 strains of Staphylococcus aureus and Staphylococcus albus from inflamed conjunctivae produced toxin causing conjunctivitis, and there was a positive correlation between the toxicity of strains and their source. Thus, the majority of staphylococci which occurred in large numbers in the initial cases of conjunctivitis and were presumed to be its cause were toxin-producers, whereas those present in insignificant numbers were generally nontoxic. 3. Positive cultures were obtained throughout the duration of the disease in 17 cases of conjunctivitis in which staphylococci were the only significant bacteria present. 4. Disappearance of the staphylococci after healing of the disease occurred in all but 3 of the 17 cases. In these 3 the bacteria persisted but were greatly reduced in number. 5. Staphylococcus toxoid [17] was effective in healing the condition in 13 of the 17 cases after failure of local treatment.

Delimitation of Staphylococcic Conjunctivitis.—The frequency of staphylococci on the inflamed conjunctiva and the difficulty of distinguishing pathogenic from nonpathogenic strains have made it difficult to delimit staphylococcic conjunctivitis. The only way of making an absolute diagnosis found in this study was to observe a case until healing occurred. A presumptive diagnosis could be made when large numbers of staphylococci of the aureus variety were present in eyes with conjunctivitis which had no other demonstrable cause, and the probability of its correctness was increased by the presence of the characteristic lesions of blepharitis or recurrent marginal ulceration of the cornea. The greatest difficulty was experienced in the evaluation of the rôle of the white staphylococci, for this study has shown that certain albus strains are capable of forming conjunctivitis-producing toxin.

Clinical Characteristics of Staphylococcic Conjunctivitis.—In the New York series there were 17 cases observed until healing occurred in which toxin-producing staphylococci were considered to be the sole cause and 37 additional cases in which staphylococci were considered to be the cause but which could not be observed to the stage of healing. Among the forms of conjunctivitis in these 54 cases were the following four clinical types: (1) conjunctivitis with an acute or a subacute onset and a definite tendency to become chronic; (2) chronic conjunctivitis with recurrent catarrhal ulcers; (3) chronic blepharoconjunctivitis, and (4) chronic catarrhal conjunctivitis without other involvement.

17. The Lederle Laboratories, New York, supplied the toxoid used in this study.

The most common form appeared to be blepharoconjunctivitis. All degrees of it were seen, from simple hyperemia of the margin of the lid to ulcerative blepharitis, which was associated in one case with sycosis vulgaris.

Staphylococcic conjunctivitis was found to have the following cardinal characteristics: (1) a protracted course, (2) resistance to local therapy and (3) frequency of concomitant blepharitis and ulceration of the margin of the cornea.

Staphylococcic conjunctivitis of the adult was found to differ markedly from the condition in the new-born (the latter has been reported separately [18]), in whom the disease was always acute or subacute, healed spontaneously or with simple antiseptic therapy and was not accompanied by involvement of the margin of the lid or by corneal involvement.

Correlation of Toxin Production of Staphylococci with Source from Which Isolated.—Staphylococci from the following sources were analyzed for the production of toxin: I. Eyes with conjunctivitis observed to healing in which staphylococci could be certainly identified as the causal factor. II. Eyes with conjunctivitis of which staphylococci were probably the cause but which could not be observed to healing. III. Eyes with conjunctivitis of which staphylococci were certainly not the sole cause but were probably a contributory cause. Included among these cases were instances of specific disease, such as trachoma, and cases of unknown etiology in which the conjunctivitis continued after disappearance of the staphylococci. IV. Eyes with conjunctivitis in which staphylococci were certainly not the cause and in which they were present in too small numbers or too irregularly to be significant.

Nonhemolytic white staphylococci were omitted from consideration in this analysis but will be discussed in a later report.

From source I, Staph. aureus was isolated in 15 cases and Staph. albus in 2. The aureus strains all produced soluble hemolysin in titers of 1:10 or greater, and all but 4 formed toxins having definite conjunctivitis-producing power. The two albus strains showed neither soluble hemolysin nor conjunctivitis-producing toxin.

From source I, Staph. aureus was isolated in 15 cases and Staph. albus in 22. All but 1 of the aureus strains showed soluble hemolysin, and 27 of the 37 showed conjunctivitis-producing toxin. Only 9 of the albus strains showed hemolysin and only 4 conjunctivitis-producing toxin.

From source III, Staph. aureus was isolated in 22 cases and Staph. albus in 24. Of the aureus strains, 16 showed soluble hemolysin and 12

18. Thygeson, P.: Tr. Am. Ophth. Soc. **34**:340, 1936.

conjunctivitis-producing toxin. Only 1 albus strain produced soluble hemolysin and only 3 conjunctivitis-producing toxin.

From source IV, Staph. aureus was not isolated, and Staph. albus was isolated in 26 cases. In none of these did Staph. albus show soluble hemolysin or conjunctivitis-producing toxin.

Thus there appeared to be a definite correlation between the source and the production of toxin, sources I and II providing a high percentage of toxin-producing strains and source IV none.

PROPERTIES OF STAPHYLOCOCCI FROM EYES WITH CONJUNCTIVITIS

The determination of the pathogenicity of a given strain of staphylococci is a time-consuming laboratory procedure requiring inoculation of animals. Many investigators have attempted to find more simple means of estimating this factor, and an apparent correlation with toxicity has been noted among the following properties of the bacterium: (1) hemolytic activity on blood agar plates, (2) production of pigment, (3) liquefaction of gelatin, (4) ability to coagulate blood plasma, (5) ability to ferment mannitol and (6) ability to produce violet growths on crystal violet agar.[19] An attempt has been made in this study to determine whether or not a correlation exists between any of these easily determined properties and the probable pathogenicity of the strain for the conjunctiva as determined by the source from which the strain was isolated and its ability to form conjunctivitis-producing and hemolytic toxins.

Staphylococci Showing Conjunctivitis-Producing Toxin.— Table 2 summarizes some of the biologic properties of staphylococci, the toxins of which were found to be conjunctivitis-producing when tested on rabbits' eyes. Their biologic activity was marked. Of the 57 strains (50 aureus and 7 albus), all but 1 produced hemolysis on rabbit blood agar plates, all but 5 produced soluble hemolysin in a titer of 1:10 or more, all but 4 coagulated human blood plasma, all but 5 fermented mannitol, and all but 9 produced a purple growth on crystal violet agar. Of the 40 strains tested for liquefaction of gelatin, 22 gave positive results.

Staphylococci Showing Soluble Hemolysin but Questionable or No Conjunctivitis-Producing Toxin.—As indicated in table 3, these strains were also active biologically. All 26 strains (22 aureus and 4 albus) produced hemolysin on plates and soluble hemolysin; all coagulated human blood plasma; all but 2 fermented mannitol, and all but 4 produced purple growths on crystal violet agar. All but 10 liquefied gelatin.

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19. Chapman, G. H., and Berens, C.: J. Bact. **29**:437, 1935.

TABLE 2.—*Properties of Staphylococci Showing Conjunctivitis-Producing Toxin* *

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
5	Aureus	++	1:1,000	+++	—	++	+	+
19	Aureus	++++	1:1,000	++++	++++	++	+	+
21	Aureus	++++	1:1,000	+++	+++	+++	+	+
28	Aureus	++++	1:100	++	—	+++	+	+
33A	Aureus	+++	1:10	++++	—	+++	+	++
34	Aureus	++++	1:10	++++	—	+++	+	++++
40A	Aureus	+++	1:1,000	++	—	—	+	++
43	Aureus	++++	1:10	++++	—	++	+	++
46	Aureus	+	1:1,000	+++	—	+++	+	++
51	Aureus	++++	1:10	+++	—	+	+	+++
54A	Aureus	+	Negative (1:10)	+++	++	+++	+	++++
56	Aureus	++	1:1,000	+++	++	+++	+	++++
63	Aureus	++++	1:1,000	++++	++++	++	+	++
72	Aureus	+++	1:10	++++	—	++	+	+
76	Aureus	++++	1:100	++++	++++	+	+	++++
84A	Aureus	++++	1:1,000	+++	++++	++++	+	+++
105	Aureus	++	1:100	++++	++++	++++	+	+
109	Aureus	++++	1:100	++++	++++	++++	+	+
113	Aureus	++++	1:100	++++	+++	++++	+	+
18	Aureus	+++	1:1,000	++++	+	++++	+	++++
68B	Aureus	++	1:100	++++	+	++++	+	+
126	Aureus	+++	1:1,000	+++	—	++++	+	+
87A	Aureus	.....	1:100	++++	+	++++	—	+
87B	Aureus	+++	1:100	+++	—	—	—	+
136	Aureus	++++	1:1,000	++++	++++	++	+	++
146	Aureus	.....	1:1,000	+++	—	+—	+	+
159	Aureus	++	1:100	++++	+++	+++	+	++++
11	Aureus	++++	1:1,000	++++	++++	+	+	++++
158A	Aureus	++++	1:100	++++	+	++	+	+++
161	Aureus	++++	1:10	++++	++++	+++	+	+
168B	Aureus	—	1:100	++++	—	++	+	+
197	Aureus	+++	1:100	+++	+++	++++	+	+
237	Aureus	+	1:10	++	—	++++	+	+++
238	Aureus	++++	1:100	+++	++++	+	+	+
239	Aureus	+++	1:100	+++	++++	+	+	+
271	Aureus	++	1:100	+++	++++	++	+	+
283	Aureus (delayed)	++++	1:1,000	+++	.....	+++	+	++
284	Aureus	++	1:1,000	++++	.....	++++	+	++++
288	Aureus	+	1:100	+++	.....	++++	+	+
290	Aureus	+	1:1,000	+++	.....	++++	+	+
291	Aureus	+++	1:1,000	++++	.....	++++	+	+
292	Aureus	+++	1:1,000	+++	.....	++++	+	++++
294	Aureus	+	1:10	—	.....	++++	+	+
295	Aureus	+	1:10	+++	.....	++++	+	+
296	Aureus	±	1:100	++	.....	++++	—	++++
301	Aureus	+	1:100	+++	.....	++++	—	+
308	Aureus	++	1:100	++++	.....	++	+	+++
311	Aureus	+	1:1,000	++++	.....	++++	+	+
335	Aureus	++++	1:1,000	++	.....	+++	—	+
341	Aureus	+	1:1,000	+++	.....	++++	+	+
32	Albus	++	Negative (1:10)	++++	—	—	—	+
45	Albus	++	Negative (1:10)	—	—	—	—	+
106	Albus	++	1:1,000	++++	—	++	+	+
81B	Albus	+	Negative (1:10)	—	—	—	—	+
289	Albus	++++	1:1,000	++++	.....	++++	+	+
322	Albus	+++	Negative (1:10)	—	.....	+	—	++
340	Albus	++	1:1,000	+	.....	++++	+	++

* The pigment was estimated after forty-eight hours' growth on plain agar. Hemolysin on plates was estimated on rabbit blood agar plates at the time of isolation of the strain after forty-eight hours of incubation. In the coagulase test, one loopful of a twenty-four hour culture on a solid medium was mixed with 0.5 cc. of citrated human plasma and incubated for three hours at 37 C. In the test for liquefaction of gelatin, the results were estimated after three weeks of incubation at 37 C. In the test for fermentation of mannitol, readings were taken after forty-eight hours. In the test with crystal violet agar, the reaction was considered positive if a violet or partly violet growth was present after thirty-six hours of incubation. The conjunctival reaction was determined on adult white rabbits. Toxin was instilled at five minute intervals for one hour, and readings were taken after twenty-four hours. + indicates a mild but definitely purulent conjunctivitis; ++++ indicates a severe purulent conjunctivitis with closure of the eye due to swelling of the lids. To eliminate faulty technic in the preparation of toxins as a factor in this test, a known toxin producer was included as a control with each group of strains studied for the production of toxin.

Staphylococci Showing Neither Soluble Hemolytic Toxin nor Conjunctivitis-Producing Toxin.—Table 4 summarizes the more important properties of the 57 strains (6 aureus and 51 albus) which produced neither soluble hemolysin nor conjunctivitis-producing toxin. They were found to be relatively inactive biologically. All hemolyzed rabbit blood agar to some degree; only 2 strains coagulated human blood plasma; only 9 fermented mannitol, and only 3 produced a purple growth on

TABLE 3.—*Properties of Staphylococci Showing Soluble Hemolysin but Questionable or no Conjunctivitis-Producing Toxin*

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
55	Aureus	++	1.10	++++	±	++++	+	—
70A	Aureus	+	1:10	++++	++++	++	+	±
78A	Aureus	++++	1:100	++++	—	++++	—	—
4	Aureus	++++	1:100	+++	++++	++++	+	—
80	Aureus	++++	1:1,000	+++	+++	+++	+	—
98	Aureus	+++	1:100	++	—	++++	+	±
112	Aureus	++++	1:1,000	++++	—	+	+	±
138	Aureus	++	1:10	++++	—	++++	—	±
117	Aureus	+++	1:10	++++	+	++++	+	—
142	Aureus	++	1:100	++++	—	++	+	—
149	Aureus	++	1:10	++++	+++	++	+	—
151	Aureus	++	1:100	++++	—	+++	+	±
173	Aureus	+++	1:100	++++	—	+++	+	±
175	Aureus	++++	1:1,000	++++	++++	++++	+	—
206	Aureus	++++	1:10	++++	+	++	+	—
232	Aureus	++	1:1,000	++++	—	—	—	—
243	Aureus	+++	1:100	+++	—	++++	+	—
250	Aureus	+++	1:100	+++	—	+	+	±
246	Aureus	++	1:1,000	++++	++++	+++	+	—
253	Aureus	+++	1:1,000	++++	—	++++	+	—
280	Aureus	+++	1:100	++++	++++	++	+	—
143	Aureus	++	1:10	++	.....	++++	+	—
50	Albus	+++	1:10	++	—	+	+	±
150	Albus	++++	1:1,000	++++	++++	++++	+	—
231	Albus	+++	1:1,000	+++	—	—	—	—
293	Albus	++	1:1,000	++++	.....	++++	+	±

crystal violet agar. Of the 52 strains tested for liquefaction of gelatin, only 6 gave positive results.

Biologic Tests as Indicators of Toxicity.—There was no absolute correlation between the production of toxin and any of the properties tested (table 5). The closest correlation was with the coagulase test: There was a correlation of 92.9 per cent between it and the production of conjunctivitis-producing toxin and of 100 per cent between it and the production of soluble hemolysin. Only 3.5 per cent of the strains which appeared to be nontoxic gave positive reactions to the coagulase test. It would thus appear that strains that gave positive reactions to the

TABLE 4.—*Properties of Staphylococci Showing Neither Soluble Hemolysin nor Conjunctivitis-Producing Toxin*

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer of 1:10	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
25A	Aureus	++	—	—	++++	—	+	—
69	Aureus	++++	—	+++	—	+++	—	—
95	Aureus (delayed)	±	—	—	—	—	—	—
66	Aureus	+÷	—	—	—	—	—	—
68A	Aureus	+	—	—	—	—	—	—
73B	Aureus (delayed)	+++	—	—	+++	—	—	—
1	Albus	÷÷	—	—	—	—	—	—
25B	Albus	÷+	—	—	—	—	—	—
27	Albus	÷	—	—	+++	—	—	—
37	Albus	+	—	—	—	—	—	—
40B	Albus	+	—	—	—	—	—	—
48	Albus	+++	—	—	—	—	—	—
52	Albus	++	—	—	—	—	—	—
57	Albus	+	—	—	—	—	—	—
58	Albus	+	—	—	—	—	—	—
59	Albus	++	—	—	—	—	—	—
62	Albus	±	—	—	—	—	—	—
64	Albus	++	—	+++	—	—	—	—
65	Albus	++	—	—	—	—	—	—
53	Albus	++	—	—	—	—	—	—
73A	Albus	+++	—	—	—	—	—	—
75	Albus	+	—	—	—	—	—	—
77	Albus	+++	—	—	—	—	—	—
78B	Albus	++++	—	—	—	—	—	—
85A	Albus	+++	—	—	—	—	—	—
85B	Albus	+++	—	—	—	—	—	—
91	Albus	++	—	—	—	—	—	—
83	Albus	++++	—	—	—	—	—	—
92	Albus	++	—	—	—	—	—	—
94	Albus	++++	—	—	—	—	—	—
107	Albus	÷++	—	—	—	—	—	—
115	Albus	++	—	—	—	—	—	—
81A	Albus	+	—	—	—	—	—	—
81B	Albus	+	—	—	—	—	—	—
116	Albus	÷	—	—	÷	—	—	—
121	Albus	+	—	—	—	—	—	—
135	Albus	++++	—	—	—	—	—	—
152	Albus	+++	—	—	—	—	—	—
153	Albus	+++	—	—	—	—	—	—
120	Albus	++	—	—	—	—	—	—
138B	Albus	+++	—	—	—	—	—	—
163	Albus	++	—	—	—	—	—	—
178	Albus	+++	—	—	—	—	—	—
221	Albus	.....	—	—	—	—	—	—
224	Albus	+++	—	—	—	÷++	—	—
234	Albus	++	—	—	—	—	—	—
241	Albus	++++	—	—	—	±	—	—
244	Albus	++++	—	—	—	÷	—	—
247	Albus	+++	—	—	—	—	—	—
266	Albus	++	—	—	÷÷÷	—	—	—
268	Albus	+++	—	—	—	—	—	—
267	Albus	++	—	—	—	—	—	—
304	Albus	++++	—	—	..	±—	—	—
318	Albus	++++	—	—	..	÷	—	—
320	Albus	+	—	—	..	÷	—	—
326	Albus	++	—	—	..	—÷	—	—
327	Albus	÷+	—	—	..	—	—	—

coagulase test would in most instances be toxic, particularly if pigment-forming. The more simple tests, those with crystal violet agar and the fermentation of mannitol, would appear to have corroborating value as indicators, even though less reliable than the coagulase test. Liquefaction of gelatin, on the other hand, was of no apparent value as an indicator of toxicity.

Is Conjunctivitis-Producing Toxin Identical with Hemolytic Toxin?—Staphylococcus toxin is known to be hemolytic, particularly for rabbit cells, to have a destructive action on leukocytes, a necrotic action when injected intradermally in rabbits, a lethal action when injected

TABLE 5.—*Correlation of Biologic Properties with Production of Toxin*

	No. of Strains	Percentage
Conjunctivitis-Producing Toxin		
Pigment	50/57	87.7
Coagulase	53/57	92.9
Fermentation of mannitol	52/57	91.2
Purple growth on crystal violet agar	48/57	84.2
Liquefaction of gelatin	22/40	55.0
Soluble Hemolysin but Questionable or No Conjunctivitis-Producing Toxin		
Pigment	22/26	86.1
Coagulase	26/26	100.0
Fermentation of mannitol	24/26	92.3
Purple growth on crystal violet agar	22/26	84.6
Liquefaction of gelatin	10/26	38.4
No Toxin		
Pigment	6/57	10.5
Coagulase	2/57	3.5
Fermentation of mannitol	9/57	15.7
Purple growth on crystal violet agar	3/57	5.2
Liquefaction of gelatin	6/52	11.5

intravenously, and a coagulating action on blood plasma. It has been a matter of some dispute as to whether these actions are the result of a single toxin or of a number of toxins.[20] Many workers, including Burnet,[21] Grosz [22] and Gengou,[23] have based their opinion that only one toxin is formed on the following facts: (1) There has been a fairly constant relationship between the hemolytic, the necrotic, and the lethal powers in different batches of toxin; (2) all manifestations of toxicity are abolished by exposure to a temperature of 56 C. for half

20. Topley, W. W. C., and Wilson, G. S.: Principles of Bacteriology and Immunity, ed. 2, Baltimore, William Wood & Company, 1936.

21. Burnet, F. M.: J. Path. & Bact. **32**:717, 1929.

22. Grosz, H.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **73**:14, 1931.

23. Gengou, O.: Ann. Inst. Pasteur **48**:135, 1932.

an hour, and (3) an antiserum prepared against the toxin neutralizes against all manifestations.

Recent findings, however, point to a multiplicity of toxins. The results of the investigations of Bigger [24] and of Glenny and Stevens [25] suggest that there are at least two hemolysins, and Dolman [26] has failed to find any parallelism between the hemolytic titers of a series of different filtrates against suspensions of red cells of different laboratory animals and concludes that the hemolysins are multiple. Panton and Valentine,[27] furthermore, have found a close relationship in different filtrates between the hemolysin and the necrotoxin but no relation between these and the leukocidin.

Topley and Wilson [20] concluded that the toxin is probably complex but that the hemolysin, the necrotoxin and the lethal toxin are all closely associated. Certain strains of staphylococci considered responsible for outbreaks of food poisoning have been found to give rise to a so-called enterotoxin which has an irritating effect on the intestinal mucous membrane of man and of the monkey. It is heat-resistant, and there seems to be little doubt that it is distinct from the other toxins.

In the present study there seemed to be evidence of a certain relationship between the hemolytic and the conjunctivitis-producing toxins, since only 5 of the 57 strains showing a conjunctivitis-producing toxin failed to give a soluble hemolysin in a titer of 1 : 10 or more. There is some evidence, however, which indicates that the two toxins may be distinct: 1. Of the 57 strains producing conjunctivitis, 5 (1 aureus and 4 albus) failed to produce demonstrable hemolysin. As recorded in table 2, one of these strains that did not produce hemolysin, no. 54 A, produced very severe conjunctivitis. 2. Twenty-six strains (table 3) gave a soluble hemolysin but no definite conjunctivitis-producing toxin. It is true that 9 of the 26 produced conjunctival reactions classed as questionable, but 4 strains (nos. 173, 175, 206 and 231), each tested twice on different animals with toxin from different batches, failed to produce conjunctivitis. 3. As can be seen in table 2, there was no strict parallelism between the potency of the conjunctivitis-producing toxin and that of the soluble hemolysin. For example, of the 10 strains giving a $++++$ conjunctival reaction, only 5 gave a soluble hemolysin in a titer of 1 : 10, while 3 gave a soluble hemolysin in a titer of 1 : 100; 1 gave a soluble hemolysin in a titer of 1 : 10, and 1 gave negative results.

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24. Bigger, J. W.: J. Path. & Bact. **36**:87, 1933.
25. Glenny, A. T., and Stevens, M. F.: J. Path. & Bact. **40**:201, 1935.
26. Dolman, C. E.: Tr. Roy. Soc. Canada (Sect V.) **26**:309, 1932.
27. Panton, P. N., and Valentine, F. C. O.: Lancet **1**:506, 1932.

Failure of Repeated Attacks of Toxin Conjunctivitis to Immunize.—No evidence was obtained to indicate that local instillations of staphylococcus toxin into the conjunctival sacs of rabbits had any immunizing effect. Thus the right eye of rabbit 136 gave a ++++ reaction on four successive courses of toxin over a period of one month, and the right eye of rabbit 139 still gave a +++ reaction after conjunctivitis had been produced in it six times during a period of seven weeks. A single drop of toxin instilled daily over a period of six weeks into the left eye of rabbit 135 failed to prevent a ++++ reaction when the usual twelve instillations given at five minute intervals were made.

Relationship of Hemolysin on Plates to Conjunctivitis-Producing Toxin.—As may be seen from examining tables 2, 3 and 4, there was no correlation between the ability of strains of staphylococci to produce hemolysis on rabbit blood agar plates and their ability to form conjunctivitis-producing toxin. Whereas all but 1 of the strains in this study produced hemolysis on blood agar plates, only 57 produced a definite conjunctivitis-producing toxin. Strain 296, for example, which produced only a very faint ring of hemolysis on the plate, caused severe conjunctivitis when its toxin was tested on the rabbit; and table 4 shows that there were 9 strains which showed maximal hemolysis on plates but for which neither soluble hemolysin nor conjunctivitis-producing toxin could be demonstrated in the filtrate.

Staphylococcus Albus as a Cause of Conjunctivitis.—While the majority of strains capable of forming conjunctivitis-producing toxin were of the aureus variety, there were 7 albus strains which produced potent toxins. It is apparent from both the clinical and the laboratory studies that certain strains of Staph. albus should be considered a cause of conjunctivitis. This question will be covered in detail in the second report of this series.

COMMENT

Staphylococcic conjunctivitis resembles diplobacillary conjunctivitis in the following important respects: (1) a protracted course, (2) failure of the bacteria to grow on or in the epithelium, (3) frequency of concomitant blepharitis, and (4) occurrence of occasional acute or subacute cases. The mechanism of production of the conjunctivitis, i. e., by the liberation of toxin in the conjunctival sac by bacteria proliferating in the secretion or on desquamated epithelial cells, is the same for both. Staphylococcic conjunctivitis, however, would seem to be much more difficult to define, owing to the frequent occurrence of saprophytic varieties of staphylococci on both normal and inflamed conjunctivae.

In addition to being the sole cause of chronic catarrhal conjunctivitis in certain cases, pathogenic staphylococci are frequently found secon-

darily complicating specific types of conjunctivitis, notably trachoma and phlyctenular conjunctivitis.

The rôle of albus strains, particularly those that produce hemolysis on rabbit blood agar, is not clear. There is no question but that some of them are active conjunctivitis-producers, but the significance of those from which no conjunctivitis-producing toxin is obtained is obscure. The possibility must be considered that some of these may produce toxin in a quantity insufficient to be measured but sufficient to produce low grade irritation of the conjunctiva.

No evidence was obtained in this study to suggest that allergy to staphylococcus toxin played any part in the reaction of the conjunctiva.

SUMMARY AND CONCLUSIONS

Evidence is advanced to indicate that toxin-producing strains of Staph. aureus and Staph. albus are pathogenic for the conjunctiva.

Staphylococci appear to be a frequent cause of chronic catarrhal conjunctivitis.

The conjunctivitis appears to be due to the liberation of a conjunctivitis-producing toxin, the staphylococci multiplying in the conjunctival secretion or on dead cells but not invading the conjunctival epithelium.

Some of the characteristics of this conjunctivitis-producing toxin are recorded. It is usually produced simultaneously with the hemolytic toxin (*a* hemolysin) but may be distinct from it.

Of the simple tests which have been proposed for estimating the toxicity of staphylococci, the coagulase test was found to give the highest correlation.

FORMATION OF DRUSEN OF THE LAMINA VITREA

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The ophthalmoscopic appearance of drusen, or "colloid" excrescences on the lamina vitrea, is well known. The changes are seen in elderly persons as numerous bright, sharply circumscribed points with slightly pigmented margins and are usually in the macular zone or around the papilla. In some cases the discrete points become confluent and rather large. The majority of patients with this condition have no visual disturbances, but when the dots are very numerous and fused, the condition can result in diminution of the visual acuity by pressure on the rods and cones that causes their atrophy. Such senile changes early attracted the attention of the pathologists, and H. Müller [1] in 1855 carefully studied them. He found that they were almost constant in persons over 60 years of age, very frequent in persons over 45 and not rare in younger persons. Histologically, they were observed to be most frequent in the neighborhood of the ora serrata and around the optic disk. Since Müller's initial study these colloid bodies have been the subject of considerable investigation, both as a senile manifestation and as a concurrence in pathologic conditions, and many theories have been offered for their origin. The present study is an attempt to review the entire subject and bring it to date, and to offer some observations which would seem to confirm the views of some of the previous investigators.

For an understanding of this condition it is first of all necessary to appreciate Bruch's membrane. This, together with Descemet's membrane and the capsule of the lens, forms a system of glassy membranes peculiar to the eye and resembling no other structures in the body. The origin of these three membranes is varied, however; Descemet's is undoubtedly derived from the mesoderm, while the capsule of the lens apparently is derived from the epithelium of the lens. Bruch's membrane, however, is more complex, and apparently both the mesoderm and the neural ectoderm contribute to its formation. There are not enough

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From the Registry of Ophthalmic Pathology of the Army Medical Museum.

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1. Müller, H.: Anatomische Beiträge zur Ophthalmologie, Arch. f. Ophth. (pt. 2) **2**:1, 1855.

embryologic observations to clarify this matter, for at the 5 mm. stage the choriocapillaris first appears, and this rapidly becomes thicker. At the 14 mm. stage it becomes separated from the outer wall of the vesicle by a clear membrane. No more than this is known of its origin in man. Wolfrum [2] has shown in investigations on animals that the basal membrane develops early, almost concurrently with formation of the secondary optic vesicle, but that the remaining layers of Bruch's membrane are formed later, concurrently with the development of the choroid. It would thus appear that the outer portion of this membrane is formed by the choroid, while the inner is derived from the neural ectoderm of the optic cup. This double origin is borne out by the finer structure of the membrane, which Salzmann [3] divided into an outer, or elastic, layer and an inner, or cuticular, layer. Sattler [4] was the first to describe the network of fibers in the outer lamella, and Smirnow [5] demonstrated that these were elastic fibers, with some collagenous fibers interspersed. The inner lamella is entirely homogeneous and forms the greatest thickness of the combined membrane. Wolfrum, however, recognized a third layer, made up of fine collagenous fibers and lying between the inner and the outer lamella, while Verhoeff and Sisson [6] expressed the belief that this third layer of connective tissue lies immediately external to the elastic lamella. The inner lamella is the true lamina vitrea. Wolfrum did not feel that this is homogeneous, but thought that it has a finely fibrillar structure and is a common product of the protoplasmic processes of the pigment epithelium and the fine endings of the collagenous fibers. For this discussion the function of the membrane is just as important as is its structure. This membrane must be easily permeable, for the rods and cones are dependent for nutrition on the choriocapillaris, from which they are separated by the membrane. That it is also permeable to cellular elements was shown by Sattler and Wolfrum, who noted leukocytes traversing it, and by Hanssen,[7] who saw red blood cells penetrating into it in the neigh-

2. Wolfrum, M.: Beiträge zur Anatomie und Histologie der Aderhaut beim Menschen und bei den höheren Wirbeltieren, Arch. f. Ophth. **67**:307, 1908.

3. Salzmann, M.: Anatomie und Histologie des menschlichen Augapfels im Normalzustande, Vienna, Franz Deuticke, 1912, p. 63.

4. Sattler: Ueber den feineren Bau der Chorioidea des Menschen, nebst Beiträgen zur pathologischen und vergleichenden Anatomie der Aderhaut, Arch. f. Ophth. (pt. 2) **22**:1, 1876.

5. Smirnow: Zum Bau der Chorioidea des erwachsenen Menschen, Arch. f. Ophth. **47**:451, 1899.

6. Verhoeff, F. H., and Sisson, R. J.: Basophilic Staining of Bruch's Membrane, Arch. Ophth. **55**:125, 1926.

7. Hanssen: Zur Frage der Glashautdrusen der Aderhaut, Klin. Monatsbl. f. Augenh. **58**:249, 1917.

borhood of hemorrhages. Axenfeld [8] stated the belief that it is possible for choroidal elements to pass through the intact membrane.

Müller found that the earliest change in the senile type of drusen is thickening of the lamina vitrea, with consequent formation of elevations of varying form and height. Smaller drusen are covered with normal-appearing pigment epithelium, but over the larger ones the epithelial coating is flattened or even lacking. The drusen are of a hyaline appearance, though Kerschbaumer [9] showed that in their earliest stages they have a finely granulated phase. Alt [10] has demonstrated that the older nodules lose their homogeneous aspect and show concentric lamination. Later still, calcium may be deposited in them, and ultimately formation of bone may occur in the mass. Isolated nodules may attain large size, or by fusion of adjacent nodules structures of bizarre shape may be produced.

Many theories have been proposed for the origin of drusen of the lamina vitrea. Donders [11] felt that they result from degeneration of the nuclei of the pigment epithelium. De Vicentiis [12] thought that degeneration of the entire pigment cell is the original cause. Leber [13] regarded the hyalin as a secretion from the pigment cells. Alt [10] expressed the view that the cells produce the hyalin as a secretion and then become hyalinized themselves. It was da Gama Pinto's [14] opinion that the cells proliferate first and that the secretion forms in the center of each clump. Kerschbaumer [9] found that the lamina vitrea of elderly persons contained granules, which became fused and formed the nodules. He stained them with eosin, fuchsin and picrocarmine, and from the reactions of the granules and the nodules he concluded that they were a hyaline secretion from the pigment epithelium. Schieck [15] was of the opinion that the pigment epithelium desquamates some of its cells,

8. Axenfeld, T.: Retinitis externa exudativa mit Knochenbildung im sehfähigen Auge, Arch. f. Ophth. **90**:452, 1915.

9. Kerschbaumer, R.: Ueber Altersveränderungen der Uvea, Arch. f. Ophth. (pt. 1) **38**:127, 1892.

10. Alt, A.: Contributions to the Pathological Anatomy of the Human Eye, Arch. Ophth. & Otol. **6**:304, 1877.

11. Donders, F. C.: Beiträge zur pathologischen Anatomie des Auges, Arch. f. Ophth. (pt. 2) **1**:106, 1855.

12. de Vicentiis: Corpi vitrei della coroidea, Jahresb. ü. d. Leistung. d. Ophth. **5**:348, 1874.

13. Leber, T.: Ueber Retinitis pigmentosa und angeborene Amaurose, Arch. f. Ophth. (pt. 3) **15**:1, 1869.

14. da Gama Pinto: Anatomische Untersuchung eines nach Critchett's Methode wegen Hornhautstaphyloma operierten Auges, Arch. f. Ophth. (pt. 1) **28**:170, 1882.

15. Schieck: Zur Genese der sogenannten Drusen der Glaslamelle, Ber. ü. d. Versamml. d. ophth. Gesellsch., 1903, p. 320.

which degenerate and then become covered with pigment epithelium. Rumschewitsch [16] expressed his belief in the theory of a cuticular secretion but thought that later the cells themselves become hyalinized. Against these views that drusen derive from the pigment epithelium there are several others that seem unlikely. Rudnew [17] considered that leukocytes can wander into the epithelium and undergo degeneration to produce these bodies. Pes [18] offered the view that scars occur in the choroid following inflammation and by contraction produce papillary projections of the choroid, which become hyalinized. Hofmann [19] believed that drusen can result from the breaking through of hyaline thrombi from the choriocapillaris. Hanssen [7] also was of the opinion that the choroid produces the drusen, basing his belief on the fact that connective tissue elements are present in many of them.

Coats [20] carefully analyzed the chief theories and has divided them into two groups. The first theory, which he called the transformation theory, is based on the transformation of the pigment epithelium cells into the hyaline masses. According to this theory, the pigment epithelium undergoes senile changes whereby the layer of cells becomes irregular, some of the cells being larger than others. The pigment granules become irregularly distributed, and either colloid globules appear in the protoplasm or the entire cell becomes converted into a colloid body. It has been offered in favor of this theory that many of the colloid bodies show no connection with the lamina vitrea, which appears to pass unchanged beneath them. Coats, however, was opposed to this view. He did not feel that the transition of the pigment cells into drusen has been sufficiently demonstrated. Also important to him was the objection that the nodules remain covered with pigment epithelium, even when they have attained considerable size. This convinced him that the excrescences originate beneath the epithelium and have elevated it, for he could not agree with the idea that the epithelium would proliferate and cover the nodules.

The second, or deposition, theory was more to his liking, and he presented a strong case in its favor. First he offered the analogy of

16. Rumschewitsch, K.: Zur pathologischen Anatomie der sogenannten Drusen der Glaslamelle der Aderhaut, Klin. Monatsbl. f. Augenh. **42**:358, 1904.

17. Rudnew: Ueber die Entstehung der sog. Glaskörper der Choroidea des menschlichen Auges und über die hyaline Degeneration der Gefässe derselben, Arch. f. Ophth. **53**:455, 1871.

18. Pes, O.: Die glasigen Körper und Papillarbildungen der Chorioidea, Arch. f. Ophth. **59**:472, 1904.

19. Hofmann: Ein Beitrag zur Kenntnis der Gefässveränderungen im Auge bei chronischer Nephritis, Arch. f. Augenh. **44**:339, 1902.

20. Coats, G.: The Structure of the Membrane of Bruch, and Its Relation to the Formation of Colloid Excrescences, Roy. London Ophth. Hosp. Rep. **16**:164, 1904-1905.

the excrescences on Descemet's membrane and the capsule of the lens, which he felt are definitely produced by the endothelial cells on the posterior corneal surface and by the cells of the lenticular epithelium, respectively. He then discussed the critical point that in many sections it has been noticed that Bruch's membrane passes beneath the drusen apparently intact. He felt that in some cases this is due to sectioning the periphery of the excrescence and thus not noticing the stalk which is connected to the membrane. However, he was convinced that the real explanation lies in the structure of Bruch's membrane, for the lamina elastica does not partake in this condition and passes unimpaired beneath the excrescences. When stained with eosin this portion seems to be the entire membrane, but when Weigert's elastic tissue stain is used it will be seen that only the outer portion of the lamina elastica is intact. The excrescence will be found to be a true bulging of the homogeneous lamina vitrea and, like it, will not stain specifically with Weigert's stain. Though Coat's conclusions are in favor of the deposition theory, he did state that both theories assume an abnormal metabolism of the pigment epithelium. He made no attempt to give the cause of this metabolic disturbance but felt that at times it is definitely pathologic, as evidenced by the occurrence of colloid bodies in diseased eyes. At other times, however, he regarded it as almost a normal change, since it is present so commonly in senile eyes.

It is my purpose in this study to present those transitional stages in the pigment epithelium which Coats felt are a weak link in the chain of argumentation in favor of the transformation theory. This evidence has been noted only in eyes which have suffered degeneration from injury or inflammation, and not in the group of senile eyes. Unquestionably the pigment epithelium during senility is subject to metabolic disturbances, and it is probably able to produce excessive amounts of the hyaline secretion, which deposits on the lamina vitrea in the form of excrescences. Consequently I shall not venture to disagree with Coats and the other proponents of the deposition theory with regard to the senile type of drusen. However, I do not feel that conditions are the same in degenerating eyes. The pigment epithelium in such eyes is subjected to a much greater noxious influence and reacts differently to produce a different type of excrescence. This does not mean that both types of drusen cannot occur in the same eye, for many eyes which contain the senile type of drusen can undergo subsequent degenerative changes.

The pigment epithelium of the retina can apparently suffer changes in its metabolism as a result of either choroidal or retinal disturbances. This is understandable when it is realized that this layer is subjected

to the cross-current of the metabolic products of the retina and the nutrient substances from the blood stream in the choriocapillaris. The earliest manifestation of the decline of this layer is the irregularity of the cells, which under normal conditions are uniformly arranged.

Fig. 1.—Swelling of isolated cells of the pigment epithelium; × 1,050.

Fig. 2.—Protrusion and beginning hyalinization of cells of the pigment epithelium; × 1,050.

Figure 1 shows isolated cells becoming swollen and pushing themselves out above the layer, though maintaining their basis on the lamina vitrea. The protoplasmic mass becomes distended, and with the dispersion of the granules of pigment the nucleus is more distinctly visible. Figure 2 illustrates the next stage, in which the cells project still farther

and are beginning to undergo hyalinization. Here it will be seen that the nucleus is becoming indistinct and that the protoplasm begins to show numerous droplets. In a case of disklike degeneration of the macula Holloway and Verhoeff [21] saw in one eye collections of swollen transparent cells filled with fine granules, which were pigmented cells that had undergone fatty degeneration, and all the transition stages in the process could be traced. In the other eye the pigment epithelium showed marked degenerative changes, with formation of numerous colloid excrescences. Along one stretch the cytoplasm and nuclei had completely disappeared, and there was a layer of colloid balls, over which there remained granules of pigment left by the pigment cells.

The response of the pigment epithelium and its subsequent changes are probably dependent on the type of baneful agent and the amount

Fig. 3.—Proliferation of the pigment epithelium, with hyalinization in the center of the clump; × 850.

of damage to the cells. These cells have the capacity of rapid proliferation and in such cases will form clumps, which may be seen protruding from the surface, as in figure 3. Since these are not healthy cells, they quickly undergo degenerative changes, and the masses are composed of a surrounding capsule of new cells with an inner core of degenerated and hyalinized older ones. The drusen can increase in size considerably in such a manner, for as the older cells degenerate new ones proliferate, increasing the bulk of the mass. This manner of growth will also account for the layer-like structure of some of the nodules, which is illustrated in figures 4 and 5. In these two figures it can also be seen that the process is not a uniform one, for some cells

21. Holloway, T. B., and Verhoeff, F. H.: Disc-Like Degeneration of the Macula, Tr. Am. Ophth. Soc. **26**:206, 1928.

incarcerated in the masses have not as yet undergone complete loss of their structure. It must be borne in mind, furthermore, that the pigment epithelium retains its secretory powers, even though its position has been changed, and that the rapidly proliferating cells are probably adding their hyaline secretion to the conglomerate. This peripheral

Fig. 4.—Hyalinized masses showing some incarcerated cells which have not yet completely degenerated; × 420.

Fig. 5.—Layers of hyaline material showing some connection to the lamina vitrea, and interspersed layer cells of proliferated pigment epithelium; × 300.

accretion is most likely the cause of some of these drusen having a concentrically layered structure, as is shown in figure 6 and, under higher magnification, in figure 7.

When the pigment cells are more severely damaged they do not have the power to proliferate, but along the entire layer there will be areas

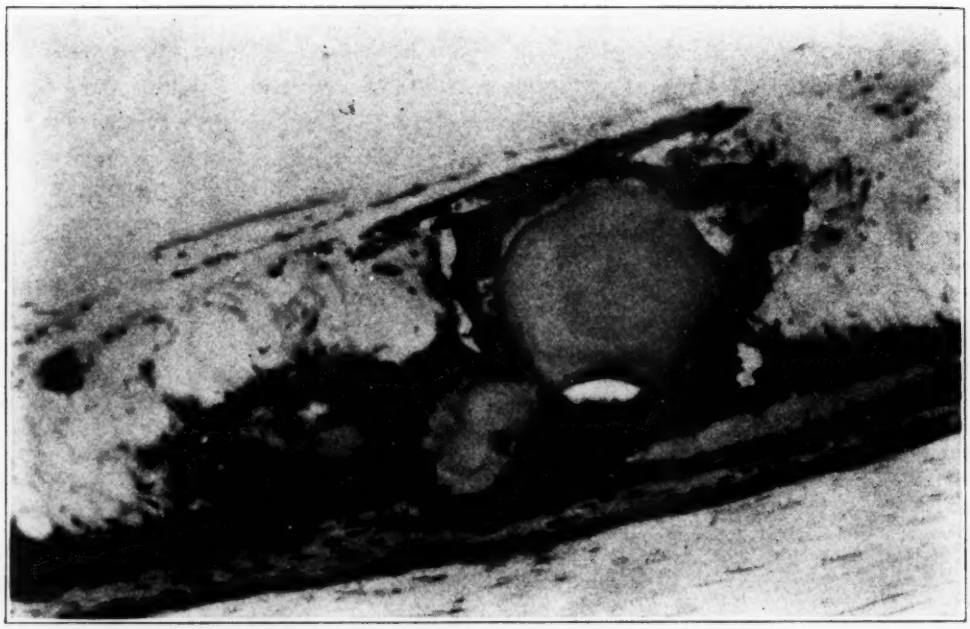

Fig. 6.—Laminated masses of hyalin completely encapsulated by proliferated pigment epithelium; × 160.

Fig. 7.—High power magnification of section of figure 6, showing details of lamination; × 915.

of lipoidal degeneration of the cells. Figure 8 shows this foamy granular mass beneath the fragmented and disarranged epithelium. Such masses have been reported in animals fed high cholesterol diets and have been cited by Heath [22] and by J. Friedenwald,[23] with the suggestion that disturbances of the lipoid metabolism are a possible cause of the formation of drusen.

Many drusen contain connective tissue elements and areas of calcification and formation of bone. From this Hanssen has assumed that the choroid alone is entirely responsible for the formation of the drusen. It is not necessary to assume this, for with inflammatory disturbances present in the choroid the lamina vitrea can be broken through and connective tissue thus invade the drusen, leading to their organization. Figure 9 shows such an area of calcification beginning in the region of the choroid and extending up into the mass. Also Holloway and Verhoeff [21] have demonstrated that connective tissue cells can be

Fig. 8.—Extensive lipoidal degeneration of the pigment epithelium forming deposits on the lamina vitrea; × 800.

derived from the pigment epithelium cells themselves, and this probably explains the frequency of the presence of these elements. Nevertheless, these authors stated that they have seen ossification of a large colloid excrescence produced by connective tissue which had entered it from the choroid.

That these colloid masses are different from the homogeneous nodules found in the senile eyes is without a doubt. As can be seen in figure 10, the senile variety of drusen shows none of the bizarre structure seen in the degenerative type, for here one has smooth homogeneous nodules encapsulated by flattened, pigmented epithelial cells and corresponding in all details to their ophthalmoscopic appearance. Granting Coat's thesis that the only valid explanation for their origin

22. Heath, P.: Ocular Lipid Histiocytosis and Allied Storage Phenomena, Arch. Ophth. **10**:342 (Sept.) 1933.

23. Friedenwald, J.: The Retinal Blood Vessels in Arteriosclerosis and Hypertension, Folia ophth. orient. **2**:175, 1936.

is the deposition theory, I do not feel that this theory can be applied to the entirely different structures seen in degenerating eyes. Many observers have attempted to differentiate the two varieties of drusen by staining methods, but these do not offer any definite evidence. It

Fig. 9.—Nodule of calcification in a hyaline mass; × 1,000.

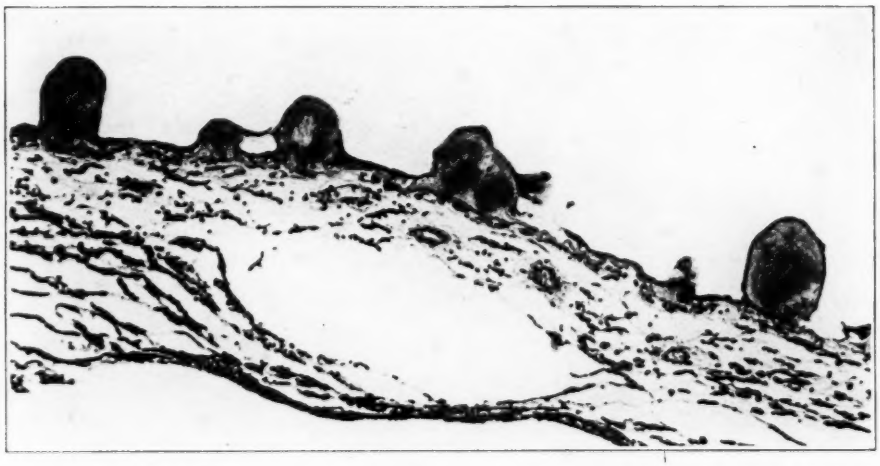

Fig. 10.—Senile type of drusen showing smooth hyaline nodules attached to the lamina vitrea and encapsulated by flattened pigment epithelium; × 205.

has been noted that with the Van Gieson stain the senile variety is yellow, while the other variety stains red. Lauber [24] has stained a

24. Lauber: The Origin of Hyaline Formations Within the Eye, Ber. ü. d. deutsch. ophth. Gesellsch. **44**:216, 1924.

number of phthisical eyes for fat. He found that where the deposits between the lamina vitrea and the pigment epithelium were stained bright red with the sudan stain, red granules could be recognized in the neighboring pigment epithelium, which he assumed to mean the presence of a chemically similar substance both in the pigment epithelium and in the deposits. I have attempted to apply the Molisch reaction for carbohydrates as a microchemical test, in order to determine whether these structures would react in the same way as Bruch's membrane, but was unsuccessful in applying this method to microscopic preparations.

Schreiber [25] has approached the problem from an experimental angle. He was indebted to Wagenmann's investigations for the knowledge that as a result of partially severing the short ciliary arteries in dogs there is a disturbance of the choroidal circulation followed by a quick degeneration of all the retinal layers, especially the rods and cones. In the neighborhood of the circulatory disturbance the pigment epithelium showed marked proliferation, with penetration of the pigment cells into the atrophic retina. Schreiber repeated these experiments and observed that numerous pigment cells had wandered completely through the retina and formed on its inner side irregular masses of cells, but that in some places they had arranged themselves in a single layer of epithelium. However, he was particularly interested in the appearance of numerous drusen-like structures. These were present not only where the epithelium lies on the lamina vitrea but also at the places to which it has migrated. Thus, they were seen on the inner surface of the retina, and because of their relationship to the pigment epithelium and their concentrically layered appearance their character could not be doubted.

In conclusion, it would therefore appear that enough evidence has been accumulated to controvert Coat's objections to the transformation theory of the formation of drusen, at least as to the formation of those drusen occurring in degenerating eyes. The transitional stages from pigment epithelium to drusen are well shown by the illustrations. The other objection—that the nodules remain covered with pigment epithelium and consequently must arise beneath it—is well answered by Schreiber's work, for he has demonstrated that, with the migration of the pigment cells into the retina and their subsequent degeneration, colloid masses are formed and are either completely or partially surrounded by pigment cells.

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25. Schreiber, L.: Ueber Drusenbildung des Pigmentepithels nach experimenteller Ciliararterien-Durchschneidung beim Kanninchen, Ber. u. d. Versamml. d. ophth. Gesellsch., 1906, p. 286.

ABSTRACT OF DISCUSSION

DR. FREDERICK H. VERHOEFF, Boston: Dr. Rones has given an excellent account of the various theories that have been advanced as to the histogenesis of drusen and has utilized specimens of his own to illustrate certain important histologic facts relating to these peculiar bodies. For convenience he classifies drusen into two main types, the senile and the degenerative, admitting, however, that each type can occur in senile eyes and also in degenerating eyes. In the senile type the substance of the body is homogeneous and hyaline in appearance, while in the degenerative type the body contains, in addition to hyaline substance, products of cellular disintegration and material such as granules and droplets of lipoid.

Dr. Rones concludes that the degenerative type results from degenerative transformation of the cells, although he assumes, inconsistently, it seems to me, that some of the hyalin is secreted by the cells concerned. He contends that his observations controvert Coats' objections to the theory of transformation. In my opinion, however, they do not throw any doubt on the validity of Coats' theory or indicate that this theory is not applicable to the degenerative type of drusen. Coats held that the inner layer of Bruch's membrane consists of a hyaline substance originally secreted by the cells of the pigment epithelium and that drusen result when certain cells later secrete an additional amount of the same substance. The question as to whether or not the hyaline substance of drusen is exactly similar to that of the cuticular layer of Bruch's membrane seems unimportant to me, since under abnormal conditions they might well differ somewhat in composition. The fact that they are both hyalin-like is sufficient. Also, it seems unimportant whether or not they are demarcated from each other.

The important question is whether or not the hyaline substance accumulates within the cells or is deposited beneath them. My own observations indicate that it is deposited beneath the cells. If it accumulates first within the cells, it should be possible at some stages to identify it there, but I have never been able to do so. The concentric lamination often seen in the larger drusen seems to me more consistent with deposition of secretion in layers than with transformation of degenerating cells.

Dr. Rones' observations do not exclude the possibility that the hyaline material found in drusen of the degenerative type is not a secretion of the cells. If it is a secretion Dr. Rones' observations do not in any way controvert Coats' theory. According to my conception, the simplest type of excrescence is due to abnormal activity of a single cell and the secretion by the cell of hyaline or colloid material beneath it which gradually pushes the cell above the surrounding surface. The body then becomes larger because secretion is added to it by the surrounding cells. When it becomes of considerable size or, in some cases, when it is still quite small, lime salts, fat and sometimes cholesterol may be deposited within it, just as in the case of many inert substances.

Under more severe degenerative conditions there are irregular proliferation and necrosis of cells and irregular formation of hyaline substance which sometimes precede and sometimes follow the severe degenerative process. In this way the various bizarre appearances referred to by Dr. Rones result.

Under certain conditions cells of the pigment layer can assume the appearance of fibroblasts and lay down a substance that cannot be distinguished from hyaline connective tissue. I believe this is similar to the substance that the pigment cells produce when they form drusen. Often a new-formed layer of pigment cells produces beneath it a cuticular membrane resembling that of Bruch's membrane.

I shall show sections of two drusen of unusual size, which illustrate some of the facts to which I have referred and also show organization of drusen partly by proliferating pigment epithelium and partly by mesoblastic cells and blood vessels derived from the choroid. They also show calcification of the drusen.

I shall demonstrate on the blackboard the relation to drusen of the fenestrated membrane in the pigment layer that I described many years ago but which still often passes unrecognized. I later found that this membrane had been previously described but not interpreted as a membrane. It is as truly a membrane as the external limiting membrane of the retina, with which it can sometimes be seen continuous at the margin of the disk.

Attention has often been called to the similarity of drusen to the so-called warts on Descemet's membrane, but I do not recall that any one has hitherto pointed to an analogy between them and the hyaline formations often to be seen in the thickened corneal epithelium in cases of bullous keratitis. I shall show a lantern slide which renders this analogy obvious. In some cases there is even produced a duplication of Bowman's membrane. Probably Bowman's membrane and the cuticular layer of Bruch's membrane are but basement membranes such as commonly occur beneath epithelium everywhere.

An interesting question is whether drusen in otherwise normal eyes are due to so-called abiotrophy or to some obscure local or general disturbance of metabolism. Certainly, as is noted by Dr. Rones, they may occur in young persons. I recall seeing them with the ophthalmoscope abundant in the fundi of a girl 15 years of age.

The pigment epithelium from many points of view has been insufficiently investigated, and I am sure that Dr. Rones' communication will stimulate interest in the study of this important structure.

Dr. Arthur J. Bedell, Albany, N. Y.: After the excellent presentation by Dr. Rones and the enlightening discussion by Dr. Verhoeff, further remarks on the pathologic problems associated with drusen seem unnecessary. I shall confine my discussion and demonstration to the thought suggested in the first paragraph of Dr. Rones' paper, the ophthalmoscopic appearance of drusen.

The fundic picture depends on the general conformation of the background, the size and number of the deposits and the degree of pigmentation.

Isolated drusen are bright yellow dots, which always lie beneath the retinal vessels. When numerous or confluent they are whiter and may form a definite layer parts of which may be thin and other portions so thick as to be almost nodular.

In cases of extensive involvement a wide zone of white spots almost completely surrounds the disk but usually avoids the region of the macula. In the cases in which the drusen are least developed the spots are discrete and more often distributed about the macula, and

occasionally they are small, brilliant and close to the nasal side of the disk. They always show a measurable degree of elevation, which is readily determined by parallactic motion or slit lamp inspection.

Macular disease may develop in an eye in which drusen are present. This confuses the picture and may lead to delay and error in diagnosis. When drusen are isolated they never cause any visual loss, and even when massed in the macular area they rarely interfere with the visual function.

There is little difficulty in making the correct diagnosis in cases in which the condition is simple and uncomplicated, but in those in which there are more intricate patterns it is well to remember that the drusen may simulate the retinal exudate found in some of the chronic inflammatory diseases of the fundus. A sheet of drusen may be mistaken for the white dots that form in the retina after the subsidence of papilledema. Careful scrutiny will, however, prove that the latter deposits are dull white and sometimes even overlie a contiguous vessel.

The white spots of so-called retinitis punctata albescens are present only when there are other evidences of retinal disease, such as peripheral pigmentation, narrow retinal vessels or, in cases in which the condition is advanced, atrophy of the optic nerve associated with pallor of the disk.

When signs of degeneration of the fundus are present and recognized, differentiation between drusen and other pathologic states is easy.

When drusen are first visible with the opthalmoscope they are small spots slightly pinker than the surrounding fundus. As they enlarge they become more yellow; then they grow paler, showing some pigmentation. The larger aggregations are often white, and not infrequently the lesion has a gray or dark brown outlining ring.

DR. BENJAMIN RONES, Washington, D. C.: With respect to Dr. Bedell's beautiful photographs, I wish to call to attention that these are not the kind of drusen that I was particularly interested in, for these are the senile variety.

Dr. Verhoeff believes that the pigment epithelium has the power to secrete the hyaline substance. This is assumed by most ophthalmologists. However, it is difficult to conceive that with such a marked disturbance in the cell itself these damaged cells have the power to produce large amounts of secretion.

It is easier to conceive that the cell itself is undergoing necrosis and hyalinization, and I attempted in these photographs to show that these damaged cells would become conglomerate and contain the pigment within them and form these layered structures.

Dr. Verhoeff has shown a layered structure of a somewhat similar nature in the corneal epithelium. However, in looking at that slide one is struck by the fact that the included cells are perfectly healthy, and the surrounding epithelium is apparently healthy, as contrasted with the pigment epithelium in these other sections, which were undoubtedly considerably damaged.

SEASONAL VARIATIONS IN THE LIPID CONTENT OF THE CRYSTALLINE LENS

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IOWA CITY

A general survey of a series of biochemical studies of human cataractous and sclerosed lenses [1] revealed distinct variations in the total lipid content at different times of the year. Such variations coincided with the four seasons. On the average, the total lipid content was low in the spring and autumn and high in the summer and winter.

Analyses of 128 lenses for determination of the total lipid content were carried out over a period of fourteen months, i.e., from March 1935 to April 1936, inclusive.

The patients from whom the lenses were extracted came from different parts of Iowa, and the operations were performed at such intervals that not a single week passed without a few lenses being analyzed. The smallest number of lenses analyzed during the course of one month (April 1935) was 11; the largest number was 23 (May and November 1935; table 1). Owing to this rather uniform distribution of material throughout the course of the study, it was possible to obtain not only monthly, but also biweekly, averages. The values are plotted in chart 1 under *c* and *b*, respectively.

Each cataract was studied and its stage of maturity determined with the biomicroscope, and later it was removed by the intracapsular method. Of the 128 lenses, 79 (61.7 per cent) had incipient cataract, 12 (9.4 per cent) intumescent cataract and 37 (28.9 per cent) mature cataract. Incipient cataract, in its biochemical make-up, is practically like the normal lens; intumescent cataract and mature cataract differ from the normal lens chiefly with respect to their water content, this being higher in the two former. There is also a slight increase in the amount of total lipids in lenses with advanced cataract and those with sclerosis. In this series of cases each monthly accumulation of lenses is represented by the three types of cataracts, i.e., incipient, intumescent and mature, in approximately the same proportion. This applies naturally also to their sclerotic conditions. There is likewise a tendency for the amount of total lipids to increase with age, which in this case varied between

From the Department of Ophthalmology, College of Medicine, State University of Iowa.

1. Salit, P. W.: Total Lipids of Human Cataractous and Sclerosed Lenses, Am. J. Ophth. **20**:157-165, 1937.

41 and 87 years. This increase was greater on the basis of absolute amounts, owing chiefly to the larger size of the older lenses, but on the basis of percentage it amounted, on an average, to less than 0.3 per year and is negligible as to its influence on the character of the curve. Moreover, the average ages of the patients from which the lenses were obtained during the course of each month did not vary considerably, and when plotted against months showed practically no correlation to the curve for the lipid content. Therefore, the variations in the lipid content at different times of the year could not be due to factors other than seasonal influences.

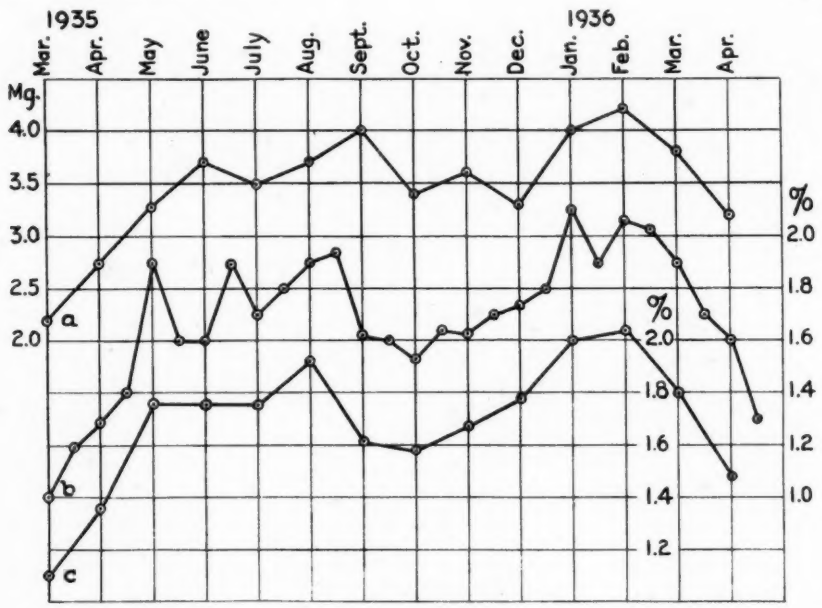

Chart 1.—Average values for the total lipid content. *a* shows the values in terms of absolute amount, or milligrams of total lipids per lens; *b* shows the biweekly values in terms of percentage, and *c*, the monthly values in terms of percentage.

Each lens was analyzed separately by a micromethod described in a previous publication.[1] In this method the lipids were extracted by solvents of fat, such as chloroform, alcohol, ether and acetone. After the evaporation of the extractives at a temperature of 65 C., the amount of lipids was estimated gravimetrically with a sensitive chemical balance.

The absolute values for lipids for the entire period of fourteen months varied between 1.2 and 6.1 mg. per lens. The values in terms of percentage varied between 0.897 and 3.142. The weights of the individual lenses varied between 0.1343 and 0.3699 Gm. The average weights of the lenses for each month, however, differed only slightly, and when plotted against months, appears to be of no significance.

The absolute value for lipids varied somewhat with the seasons of the year (table 1 and chart 1, *a*): they were lowest in the spring, increased somewhat during the summer, then decreased slightly during the fall and reached their maximum in the winter. On the basis of percentage, the seasonal variations were much more pronounced (table 1, and chart 1, *b* and *c*). The average value for March 1935 was 1.10 per cent (chart 1, *c*). There was then a steady increase throughout the succeeding months until a value of 1.92 per cent was reached in August. This means an increase of nearly 73 per cent over the value for March. Then the value decreased, reaching a relatively low level of 1.59 per

TABLE 1.—*Seasonal Variations in the Total Lipid Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Total Lipids per Lens, Mg.	Average Amount of Total Lipids, Percentage
1935					
March	15	63.6	0.203	2.3	1.10
April	11	69.7	0.194	2.8	1.35
May	23	70.7	0.193	3.3	1.75
June	19	73.2	0.209	3.7	1.75
July	12	63.6	0.193	3.5	1.75
August	14	69.6	0.205	3.7	1.92
September	13	67.8	0.210	4.0	1.62
October	15	69.2	0.213	3.4	1.59
November	23	69.6	0.214	3.6	1.67
December	15	61.3	0.189	3.3	1.77
1936					
January	21	63.7	0.192	4.0	2.00
February	13	68.3	0.209	4.2	2.04
March	17	67.5	0.208	3.7	1.80
April	17	69.2	0.212	3.2	1.47

cent in October—a level indicating a drop of nearly 21 per cent as compared with the value for August but more than 43 per cent higher than that for March of the same year. A second high peak, of 2 per cent, was reached in January 1936, and a peak of 2.04 per cent was reached in February 1936, which is almost double the value for March of the preceding year and more than 26 per cent higher than that for October 1935. The value then decreased rather rapidly, until by April 1936 it reached 1.47 per cent, which is more than 8 per cent lower than the value for October of the previous year.

The graph based on monthly averages (chart 1, *c*) has a rather smooth curve, showing that these variations are not merely chance but are the result of profound systematic and periodic changes in physiologic processes.

Previous to the analysis of the total lipid content in the crystalline lenses, similar analyses had been carried out for determination of the cholesterol and phospholipid contents.[2] The cholesterol content was determined by the colorimetric method of Bloor.[3] For the estimation of the phospholipid content a modification of Whitehorn's [4] colorimetric method was employed. The ages of the patients whose lenses were analyzed varied between 45 and 85 years in the cases in which the cholesterol content was determined and between 41 and 87 years in the cases in which the phospholipid content was determined. Unfortunately, the number of lenses analyzed each month was very small in both series of cases, not quite half that in the cases in which the total lipid content was determined, but the analysis for the cholesterol content extended

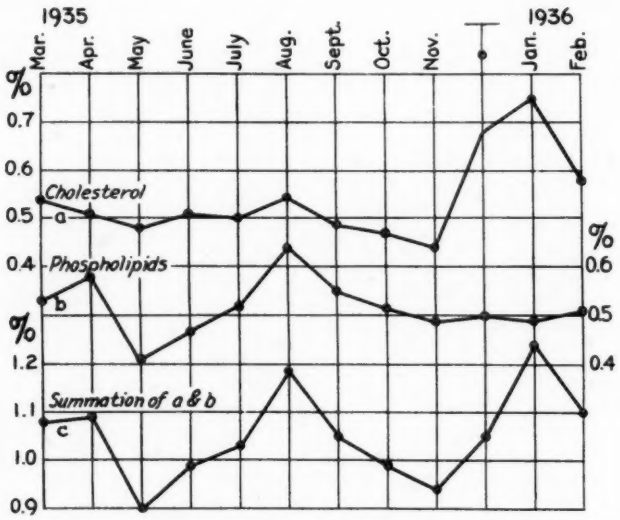

Chart 2.—Average values for the cholesterol and phospholipid contents in terms of percentage.

over fifteen months and that for the phospholipid content over sixteen months, as compared with fourteen months for the determination of the total lipid content. In order to obtain more reliable averages the differ-

2. (*a*) Salit, P. W., and O'Brien, C. S.: Cholesterol Content of Cataractous Human Lenses, Arch. Ophth. **13**:227-237, 1935. (*b*) Salit, P. W.: Phospholipid Content of Cataractous Human Lenses, Brit. J. Ophth. **19**:663-671, 1935; (*c*) Phospholipid Content of Cataractous and Sclerosed Human Lenses: A Biochemical Study of Lenticular Changes, Arch. Ophth. **16**:271-283 (Aug.) 1936.

3. Bloor, W. R.: Studies on Blood Fat: II. Fat Absorption and the Blood Lipoids, J. Biol. Chem. **23**:317-326, 1915; The Determination of Small Amounts of Lipid in Blood Plasma, ibid. **77**:53-73, 1928.

4. Whitehorn, J. C.: A Method for the Determination of Lipoid Phosphorus in Blood and Plasma. J. Biol. Chem. **62**:133-138. 1924. Salit.[2c]

ent values for identical months were pooled, and curves were obtained for only one year, i. e., twelve months. This was first done for the values for cholesterol and the phospholipids separately (chart 2, *a* and *b*, and chart 3, *a* and *b*). A third curve was obtained by the addition of these two (chart 2, *c*, and chart 3, *c*). On account of the small number of lenses, no great accuracy can be claimed for these curves. They do, however, bear the character of certain definite periodic variations or tendencies. The influence of the factor of age was operative here even less than in the case of the total lipid content, although there are two high points in each curve coinciding with rather high averages for age; the influences of the latter may account for only an exceedingly small part of the high values. Similarly, the fact that some of the lenses

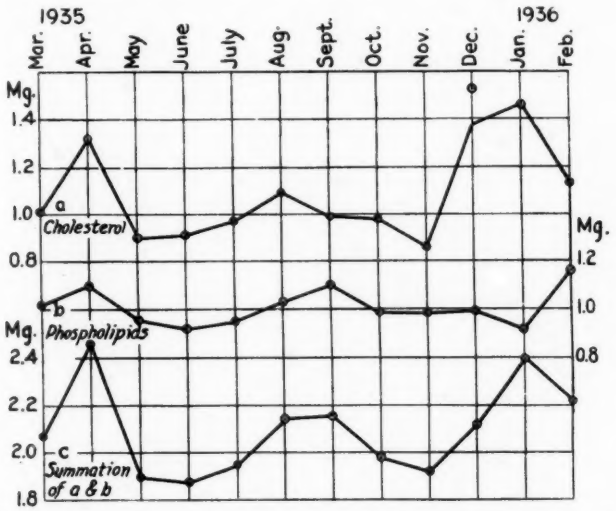

Chart 3.—Average values for the cholesterol and phospholipid contents in terms of absolute amount, or milligrams per lens.

had a mature cataract has no bearing on the nature of the curves for the same reason that was pointed out in connection with the total lipid content. The variations, therefore, as in the case of the total lipid content, must be attributed to seasonal influences alone. In the curves showing the values in percentage (chart 2, *a* and *b*) both the cholesterol content and the phospholipid content are seen to be low in May. They increased during the early months of summer, reaching a high level in August; this increase was especially pronounced in the phospholipid content. This was followed in both cases by another decline, the lowest level being reached in November. In December only 1 lens was analyzed for the cholesterol content, and it happened to be the one with the highest cholesterol content. As a single value this content has no significance,

but in conjunction with the cholesterol content of the 3 lenses analyzed in January, which likewise happened to be of a relatively high cholesterol content, it indicates a second and a more pronounced increase in the cholesterol content after the onset of winter, the highest level being

TABLE 2.—*Seasonal Variations in the Cholesterol Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Cholesterol per Lens, Gm.	Average Amount of Cholesterol, Percentage
March	6	66.0	0.201	1.04	0.54
April	10	63.6	0.196	1.34	0.50
May	9	66.1	0.198	0.93	0.48
June	3	69.0	0.189	0.93	0.51
July	6	70.1	0.205	0.99	0.50
August	16	65.6	0.206	1.11	0.54
September	18	70.7	0.222	1.04	0.48
October	18	62.4	0.215	0.99	0.46
November	9	64.1	0.204	0.89	0.44
December	1	79.0	0.186	1.54	0.83
January	3	74.0	0.207	1.47	0.74
February	6	61.8	0.203	1.15	0.58

TABLE 3.—*Seasonal Variations in the Phospholipid Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Phospholipid per Lens, Mg.	Average Amount of Phospholipid, Percentage
March	5	60.[illegible]		1.04	0.53
April	9	70.9	[illegible]09	1.12	0.58
May	6	69.2	0.204	0.97	0.41
June	3	68.3	0.205	0.94	0.47
July	3	62.0	0.196	0.96	0.52
August	2	71.0	0.163	1.04	0.64
September	12	[illegible]0	0.206	1.10	0.55
October	7	69.5	0.193	0.99	0.52
November	16	69.6	0.195	0.95	0.49
December	12	68.8	0.203	1.01	0.51
January	9	68.2	0.192	0.92	0.49
February	17	63.8	0.220	1.08	0.50

reached during December and January. A lower cholesterol content was registered during February, yet this value was still considerably higher than any of the values obtained during the spring, summer and fall. On the other hand, there were practically no further changes in the phospholipid content after November throughout the winter.

By summation of the two curves a third curve (chart 2, *c*) was obtained, which strikingly resembles the one for the total lipid content; this shows that the low levels were in May and November, and the high points coincide with August and January.

When the corresponding absolute values for cholesterol and the phospholipids are plotted against the twelve months of the year (chart 3, *a* and *b*), they resemble the ones based on percentage. The seasonal variations in the phospholipid content, however, are shown to be not as pronounced as those obtained on the basis of percentage, but the variations in the absolute amounts in the curve for cholesterol are seen to be even more definite than those in the curve obtained on the basis of percentage. By summation of the curves for absolute amounts of cholesterol and that for absolute amounts of phospholipids, a third curve (chart 3, *c*) was obtained which resembles closely the summated curve obtained on the basis of percentage and consequently also the curve for the total lipid content.

The total lipids of the crystalline lenses consist of approximately equal amounts of cholesterol, phospholipids and ordinary fat.

From the foregoing statements it seems certain that the lipid content of crystalline human lenses is subject to seasonal variations; these variations are chiefly due to changes in the cholesterol and the phospholipid content.

SUMMARY

A total of 128 human crystalline lenses were analyzed for the total lipid content over a period of fourteen consecutive months. The lenses, which consisted of 79 with incipient cataract, 12 with intumescent cataract and 37 with mature cataract, were removed from the eye in toto by the intracapsular method. The lipids of each lens were extracted separately by the use of such solvents of fat as chloroform, alcohol, ether and acetone, and their amounts were estimated gravimetrically with a sensitive chemical balance.

By plotting the monthly averages of lipids, both in terms of percentage and in terms of absolute amount, a characteristic, four-phasic curve was obtained, in which the two crests coincide with summer and winter and the two troughs coincide with spring and fall. Since nearly 62 per cent of the lenses had incipient cataract or were practically normal and the differences in the composition of the lipids of mature cataract as compared with normal lenses consist only in a slightly increased total lipid content and a reduced phospholipid content, these striking variations as represented by the curve must be attributed solely to seasonal influences.

Two groups of similar lenses had been previously analyzed for the cholesterol content and the phospholipid content, respectively. By plot-

ting their values in the same way, curves were obtained of a more or less diphasic nature but of opposite tendencies; the curve for the phospholipid content shows a pronounced increase in the content during the summer and a relatively low value during the rest of the year, whereas the curve for cholesterol content has its highest elevation during the winter and its lowest levels during the rest of the year. When the two curves were summated a third curve was obtained which strikingly resembles that for the total lipid content, the level for the phospholipid content representing the elevated part during the summer, that for the cholesterol content representing the elevated part during the winter and the low levels coinciding with the spring and autumn, exactly as in the curve for the total lipid content.

ABNORMAL OCULAR AND PUPILLARY MOVEMENTS FOLLOWING OCULOMOTOR PARALYSIS

REPORT OF A CASE

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On recovering from palsy of the oculomotor nerve, the formerly paralyzed ocular muscles may make movements which are contrary to or incoordinate with normal associated motions of the globes and eyelids. For example, in looking down, the upper eyelid, which normally follows the globe in its vertical movements, may not descend. Indeed, the eyelid may even retract upward when the globe moves downward. Another atypical ocular movement abnormally associated with downward gaze is inward rotation of the globe. A third and more rare type of abnormal movement associated with looking down is constriction of a pupil which at rest is dilated and inactive to light or in accommodation. In the following case report all three types of abnormal movements occurring simultaneously in one eye are described.

REPORT OF CASE

J. D., a man aged 28, applied to the dispensary of the Lincoln Hospital for treatment of a contused lip. On examination it was noted that when the patient looked down the left upper eyelid moved upward, the eyeball moved slightly inward, and the pupil constricted. When questioned as to his ocular condition, the patient replied that he was treated in the Bellevue Hospital in 1930 for headache and closure of the left eye.

The history recorded in the Bellevue Hospital is that on July 2, 1930, while running through a rain storm, he felt a sudden pain in the nape of the neck. When he reached home three minutes later, he collapsed and became semiconscious. Later in the afternoon he became nauseated. In the evening he complained of severe occipital headache. On the following morning the headache settled over the left eyebrow. For the next few days, in addition to headache and drowsiness, there was vomiting. On July 8 it was noted that the left eyelid began to droop. Two days later the drooping of the lid became complete; he then entered the hospital.

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Examination revealed the patient to be slightly stuporous, although cooperative. The significant findings were oculomotor ophthalmoplegia in the left eye with complete ptosis, external strabismus, paralysis of mesial and vertical movements of the globe, and a dilated pupil, which was fixed to light and in accommodation. The fundi were normal. There was a questionably positive Babinski sign on the right. The rest of the examination, including a complete test of the neurologic status, gave negative results.

Throughout the patient's stay in the hospital his temperature was normal. The pulse rate was slow, ranging between 54 and 64 beats per minute. The blood pressure was 120 mm. of mercury systolic and 65 mm. diastolic. Lumbar puncture performed on the day after admission revealed xanthochromic fluid which contained many crenated red blood cells. A second puncture, made three days later, showed bloody spinal fluid with supernatant xanthochromic fluid. The Wassermann tests of the blood and spinal fluid were negative. Cultures of the spinal fluid gave negative results. The blood count showed 9,100 white blood cells, with 72 per cent polymorphonuclear cells, 26 per cent lymphocytes and 2 per cent eosinophils. The urine was normal; there was no sugar or albumin.

Under observation the patient was drowsy, and he vomited on several occasions. There was slight and gradual improvement in his condition. At the end of two weeks' residence he felt well enough to leave the hospital against advice. The clinical diagnosis was subarachnoid hemorrhage due to a ruptured aneurysm. The subsequent history, as obtained from the patient, was that six weeks after he left the hospital the drooping eyelid began to open and since then he has had double vision.

Examination made at the dispensary of the Lincoln Hospital on Feb. 13, 1937, revealed oculomotor ophthalmoparesis, with abnormal movements of the eyelid, globe and iris of the left eye. When the patient was asked to look down the following phenomena were noted to occur in the left eye: (*a*) The superior eyelid retracted slightly upward. (*b*) The eyeball rotated slightly inward. (*c*) The pupil, which was 6.5 mm. in diameter and inactive to stimulation by direct or indirect light or in accommodation, contracted to 3 mm. The constriction of the pupil was slow and appeared one or two seconds after the patient began to look down.

Inward motions of the left eyeball as part of conjugate movements of the eyes to the right also produced retraction of the upper eyelid. No motion was noted in the upper eyelid or in the iris when the eyes gazed upward; the only abnormal movement noted in this position was slight internal rotation of the globe. Apparent ptosis of the left eyelid was present when the patient looked up. Vertical movements of the left eyeball were much impaired and more so for upward than for downward motion. The power of convergence was lost. Conjugate movements to the left were normal.

The patient was able to shut both eyes. During this procedure the right eyeball turned upward, whereas the left eyeball turned outward and slightly upward. Associated with the closure of the lids there was a slight constriction of the pupil in each eye. The corneal reflexes were normal.

When the patient was asked to look down, with the eyes initially closed, the left superior eyelid snapped upward.

All the ocular movements and reflexes in the right eye were normal. The optic fundi were normal. The visual fields when tested grossly showed no defects in any of the quadrants.

The deep tendon reflexes were slightly increased on the right. There was no Babinski sign. The gait, coordination, muscular power, and sensation, and the function of the cranial nerves other than that of those supplying the left eye were normal. There were no abnormal movements of the extremities, no nystagmus and no gross mental disturbances. Clinical examination of the thoracic and abdominal viscera gave negative results.

COMMENT

The simultaneous occurrence of retraction of the upper eyelid, inward rotation of the globe and constriction of the pupil in a paretic eye is not a frequent observation. The delayed and slow constrictions of an otherwise immobile pupil, in association with downward gaze or, less conspicuously, in association with closing of the eyes, are rare phenomena.

The failure of the superior eyelid in a formerly ophthalmoplegic eye to droop when the gaze is directed downward has been known as the pseudo-Graefe phenomenon.[1] This, however, is a misleading eponym which has crept into the ophthalmologic literature because the mechanism of the phenomenon was not clearly understood. There is no relation in mechanism or etiology between the lag of the lid in cases of exophthalmic goiter and that seen after recovery from oculomotor palsy. In the latter instance not only does the lid lag but it actually retracts, an effect due to contraction of the levator muscle, just as the internal rotation of the globe is due to contraction of the internal rectus muscle. These contractions and constriction of the pupil are the result of indiscriminate regeneration of oculomotor nerve fibers. During the process of healing, many of the regenerated axis-cylinders enter the empty nerve sheaths, passing to muscles other than those which they originally supplied. The net result is that nerve impulses meant predominantly for one muscle are shunted to all muscles innervated by the regenerated nerve, so that all the muscles contract simultaneously. Sometimes the axis-cylinders of one branch regenerate only in paths to one or two other muscles, so that the contractions en masse are not seen.

The problem of nerve regeneration and recovery of function of transected oculomotor nerves has been investigated by one of us (M. B. B.) in collaboration with Dr. Fulton at Yale University. Complete results will be published at a later date. In brief, it was found that in every monkey or chimpanzee in which the oculomotor nerve was cut

1. Bender, M. B.: The Nerve Supply to the Orbicularis Muscle and the Physiology of Movements of the Upper Eyelid, Arch. Ophth. **15**:21 (Jan.) 1936. Bielschowsky, A., in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1907, vol. 8, pt. 2, chap. 11, p. 202; Lectures on Motor Anomalies of the Eye: II. Paralysis of Individual Eye Muscles, Arch. Ophth. **13**:33 (Jan.) 1935.

regeneration had occurred after a period of several months. Retraction of the upper eyelid and inward rotation of the globe were present whenever the normal eye looked downward, outward or, sometimes, upward. Vertical movements of the globe as part of mass contraction of all the ocular muscles were not seen, because the inferior oblique, the inferior rectus and the superior oblique muscle are antagonistic. When these muscles contract simultaneously, no gross movements result. Thus far, abnormally associated movements of the paralytic sphincter pupillae have not been observed in the experimental animal.

Section of the regenerated oculomotor nerve abolished all the abnormal movements in these animals, and the ophthalmoplegia reappeared. Analogous to observations made on regenerated oculomotor nerves were those made after experimental section of facial nerves. The results were almost identical. All experimental data seem to support the theory that the abnormal mass movements which occur after section of nerves are the result of faulty peripheral nerve regeneration and are not due to faulty nuclear regeneration as suggested by Fuchs.[2]

SUMMARY

A case is described in which a patient who had recovered from oculomotor ophthalmoplegia showed abnormal movements in the upper eyelid, the internal rectus muscle and the pupil. All three abnormal movements occurred simultaneously and in association with downward gaze. These movements are similar to those found after experimental section of oculomotor nerves in monkeys and chimpanzees. The abnormal movements are due to faulty peripheral nerve regeneration.

2. Fuchs, E.: Association von Lidbewegung wit seitlichen Bewegungen des Auges, Beitr. z. Augenh., 1893, no. 11, p. 12.

SOME NEW CONCEPTIONS REGARDING EGOCENTRIC VISUAL LOCALIZATION

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The fact that one always sees things definitely placed in space in relation to oneself is so obvious that one rarely considers the mechanism of the appreciation of the spatial positions. More rarely does one consider the fact that such appreciation of position is a purely subjective phenomenon. This is occasionally impressed on one by discrepancies between the position of an object as determined by the eyes and that determined by tactile-proprioceptive sensation or objectively determined position. This discrepancy appears in cases of recent or fresh paralysis of the extra-ocular muscles which causes binocular diplopia. Immediately after the paralysis there is confusion because of the discrepancy, followed by gradual readjustment as the subject learns to disregard the falsely localized image. The objective interpretation of the relative positions of the double images in such cases, based on the simple laws of monocular projection, is well known, but analysis of the subjective phenomena involved has been neglected, primarily because of an inadequate understanding of the subjective aspects of localization under normal conditions of ocular function. Similarly, there has been a lack of understanding of the subjective phenomena of so-called anomalous correspondence of squints which has led to many erroneous conceptions in an attempt to explain the anomaly. It is the purpose of this paper to lay the foundation for a more correct interpretation of these phenomena by an analysis of subjective egocentric localization under normal conditions. An analysis and interpretation of the sensory anomaly in squints will be presented in a later publication.

Subjective egocentric, or absolute, localization (von Graefe's "subjective orientation") is perception of the positions of seen objects in relation to the observer's body image. The term may be used also to denote the visually perceived positions of objects in relation to the body image. The body image is the instantaneous subjective spatial representation of the observer's own body given as a composite of the representations by the various sensory systems, most of the parts visible to the observer himself being represented visually while the nonvisible parts are represented by tactile, proprioceptive and gravistatic sensa-

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tions. The correct fitting together of these representations depends on subjective correlation of the various sensory systems.

A characteristic of vision is that a stimulus affecting the retina is not perceived as stimulation of a definite part of the body but is perceptually externalized and is identified directly with its apparent source. This externalized representation of the stimulus is localized subjectively with respect to the body image, especially the head portion, without particular reference to, or consciousness of, the exact anatomic site of the stimulation. The retina, therefore, cannot be said to have a local sign as a part of the head image but has instead a subjectively externalized intrinsic local sign which is spatially correlated with the head image.

What little evidence there is points to an innate origin of this spatial correlation between the externalized visual representations and the head image rather than its acquisition through individual experience. Young infants, before they are able to grasp seen objects, turn the head or eyes toward a source of light once the latter has been "found" by the fovea, which must indicate that the source is localized subjectively, at least in a crude fashion, by foveal fixation. Foveal fixation and thus probably foveal localization are present before localization develops in the periphery of the visual fields. The so-called developmental unfolding of the visual field postnatally, as evidenced by the infant turning its eyes or its head toward a peripheral source of light, is probably the development of subjective localization of the periphery rather than the actual beginning of the perception of stimuli by the peripheral visual elements. The fact that simple awareness of stimuli in the peripheral field appears to be a more highly developed function than accuracy of localization suggests that the former is present fundamentally. Of course, in the infant there is no definite indication of simple awareness of particular stimuli except in behavior dependent on localization.

In normal binocular vision the two eyes seem to function subjectively as a single organ. That is, one is not aware of a visual stimulus affecting two separate organs, since only a single source is seen. Binocular vision is generally conceded to be an innate faculty. The singleness of binocular vision is singleness of externalized representation. Verhoeff [1] gave the name "unification" to this and explained it by his theory of replacement, according to which the unified binocular percept is a composite, resembling a mosaic, of the respective monocularly seen elements. Thus the percepts arising from corresponding points are not seen simultaneously, but one replaces the other. Which of the two is consciously seen is determined by their relative attention

1. Verhoeff, F. H.: A New Theory of Binocular Vision, Arch. Ophth. **13:** 151-175 (Feb.) 1935.

values; the one which arouses attention of the higher degree is given precedence. A fundamental for such a theory is that percepts arising from corresponding points always are localized in the same spatial position with respect to the head image; otherwise replacement would result in incongruities. In fact, this is the subjective basis for the conception of correspondence. Thus, the representations of corresponding points are interchangeable without subjective alteration of the spatial nature of the binocularly unified image.

In agreement with the theory of replacement, therefore, normal binocular visual localization may be conceived to be the result of the proper fitting of the separate, monocularly originating elements into a common spatial scheme or framework, the localization of which is innately correlated with the body image, especially the head image, so as to agree with that given by the other senses. The spatial representations of corresponding retinal points may be said to stand in a mutual relationship to a single subjective localization. This will be considered more fully later.

Subjects with normal binocular vision and without deviation of the visual axis of either eye under cover will see objects of foveal fixation in the same position monocularly, when one eye is covered but not closed, as binocularly. If, however, there is lateral deviation of the axis of the eye which is covered, the localization of objects seen monocularly will appear displaced, relative to their binocular localization, in the direction of deviation of the axis of the covered eye. A similar phenomenon is associated with change of fixation from near to distant objects, and vice versa. When, for instance, one eye is covered but not closed and the other eye is fixed first on a distant object, and then changes fixation to a near object which is alined monocularly with the distant one, the whole monocularly seen field will appear to shift its position toward the side of the uncovered eye. This depends on the change in convergence of the visual axes which is associated with change of fixation from distant to near objects. If instead of simply being covered one eye is closed voluntarily with the lids, these shifts in localization of the monocularly seen field either do not occur or are of slight, variable degree. One intentionally can vary these phenomena by controlling the position of the nonobserving eye whether it is covered or closed, particularly in the former case if there is enough phoria in the proper direction to maintain the position of the covered eye.

In these phenomena the subjective localization of the seen field is obviously influenced by the actual direction of the visual axis of the covered eye. This leads to the conclusion that whether an actual observation is made monocularly or binocularly, as long as both eyes are

open the individual positions of the two foveal visual axes are simultaneously and jointly associated with a single definite subjective localization.

Association of stimulation of corresponding points with a single subjective localization, therefore, makes it unnecessary to distinguish consciously between the respective monocularly seen parts of a binocularly unified image, and also, by virtue of replacement, makes it impossible to tell under ordinary circumstances whether an object is imaged on only one retinal point or simultaneously on both of a pair of corresponding retinal points. The fixation intent is normally directed toward binocular fixation and is therefore subjectively associated with simultaneous foveal fixation in both eyes. Consequently, regardless of whether an object is imaged on one or on both foveae, it is localized subjectively as if imaged on both, and the localization is that which is associated with the instantaneous positions of both foveal visual axes.

The phenomena associated with paralysis of the extra-ocular muscles and with passive rotation of the eyeball indicate that the actual position of each foveal visual axis is correlated with a definite innervational status of the extra-ocular muscles which effect the rotational position of the eye. For example, after paralysis of the lateral rectus muscle, attempts to rotate the affected eye outward are at first attended by a subjective shift of the whole field seen by this eye in the direction of, and by an amount approximately equal to, that of the intended transfer of foveal fixation. Thus the objects imaged by the fovea are localized subjectively in the position of the intended foveal fixation as represented by the muscular innervation which would normally accomplish it.

In the case of passive rotation of the eyeball, by external means or as occurs reflexly in Bell's phenomenon, the objects imaged by the fovea, and the rest of the field, according to its local sign arrangement about the former, are seen in the localization given by the intended innervation of the extra-ocular musculature. Since this is unchanged during passive rotation, the succession of objects imaged by the fovea is seen as a succession of objects moving through the given localization in a direction opposite to that of the displacement of the foveal visual axis. Or, since the field is seen as a whole, it appears to move as a whole in this direction.

One is conscious not of the actual positions of the foveal visual axes as such but only of the single subjective localization with which they are jointly associated. Their individual positions both enter into the determination of this localization, and for this purpose it is apparently necessary for a conscious image to be obtained from only one fovea. The innervational status of each extra-ocular musculature, then, must be the means of subjectively, but not consciously, representing, in

physiologic terms, the position of its foveal visual axis. According to this, regardless of the actual position of the foveal visual axis, the position which enters into the subjective localization is that which the innervational status of the extra-ocular musculature represents as present.

The localization with which we are concerned is essentially a gross one with relation to the body image. It involves in a similarly gross fashion depth perception, that is, the distance of seen objects from the body image, especially the distance of the object of fixation. It thus utilizes primarily the same factors as monocular depth perception, since only the localization with which corresponding points are correlated is involved, and binocular parallax therefore does not serve it directly. The latter, however, may furnish clues to the size-to-distance relationship and thus indirectly aid in the subjective localization.

Certain phenomena indicate that subjective representation of the relative positions of the foveal visual axes is a factor in subjective egocentric distance localization. The phenomena mentioned consist of changes in the apparent size of objects on inducing so-called passive convergence or divergence as by slipping prisms before the eyes. Passive convergence results in an apparent diminution of size and consequently a judgment of remoteness. Passive divergence causes the reverse phenomenon. As was pointed out by Verhoeff,[2] the only logical interpretation of these phenomena is that the subjective representations of the positions of the visual axes are associated directly with the idea of size and through this with the idea of distance. Since the visual angle subtended by the object actually remains constant, the apparent size of the object is that which, for this visual angle, is correlated with the instantaneous innervational statuses of the extra-ocular musculatures representing the positions of the foveal visual axes.

On considering these phenomena, it is apparent that one does not see distance or intervening space as such but sees only the apparent sources of one's retinal images. One's impression of visual space is thus directly dependent on, and is in terms of, one's perceptions of the spatial nature of its contents, which invariably exist for one in three dimensions. This means not that one always localizes the objects seen correctly, that is, in agreement with objective localization in three dimensions, but that one's idea of the spatial existence of seen objects is always three-dimensional, whether or not one can express it accurately in objective terms. Seeing something always implies seeing it somewhere in a three-dimensional spatial relationship to oneself, whether it is observed with one eye or with both eyes.

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2. Verhoeff, F. H.: A Theory of Binocular Perspective, Am. J. Physiol. Optics **6**:416-448, 1925.

In normal binocular visual localization, then, the following fundamental correlations are present: (1) the correlation between the fixation intent and simultaneous binocular foveal fixation; (2) the correlation between the position of each foveal (fixation) visual axis and the innervational status of the respective extra-ocular musculature, this position being represented subjectively in terms of the innervational status; (3) the simultaneous, joint correlation between the positions of the two foveal visual axes, as given in these terms, and a single subjective localization in three dimensions, the third dimension, or depth, involving (4) a further correlation between these subjective innervational representations and the size, and thus the distance, of seen objects. By virtue of the consistent orientation of other points in each field about the fixation point in accordance with their local signs, these correlations may be extended to corresponding points in general. I subscribe to the theory of an innate origin of these fundamental correlations on the basis of phylogenetically acquired cerebral nervous connections. The rationale for this belief lies in the analogy to other phylogenetic changes in the central nervous system which serve binocular vision. These are the connections of the motor nerves serving conjugate and disjunctive movements and the partial decussation of the optic nerve fibers which serves the overlapping of homonymous halves of the visual fields.

RETINAL CORRESPONDENCE

One may now examine more critically the relations generally existing between so-called corresponding points. As was emphasized previously, normal correspondence between retinal points in the two eyes requires that percepts, arising from these points, always be localized interchangeably in the same spatial position in relation to the head image. Correspondence thus means a fixed mutual association with a single subjective localization in three dimensions. Its usual definition in terms of subjective direction, whether referred to the so-called cyclopean center of directions or any other point in the head image, is inadequate, since the term direction involves only two dimensions. Moreover, to say that normally corresponding points are those points represented subjectively by identical directions is incorrect, for, in the first place, a single retinal point is represented subjectively not by a direction but by a spatial position in three dimensions as long as one has cognition of three-dimensional space, and, in the second place, any retinal point in one eye, when the other eye is not closed consciously, is not normally correlated independently with an individual subjective localization, even though the other eye is completely excluded from the observation. As was stated previously, there is normally a simultaneous, joint correlation between the subjective representations

of the positions of the foveal (fixation) visual axes and a single subjective localization in three dimensions. Likewise, the representations of the visual axes of any other pair of corresponding points are, through their local sign relations to their respective fixation axes, similarly correlated with a single subjective localization. These representations of the visual axes, however, cannot be said to indicate independently individual subjective localizations. They are unconscious representations in physiologic terms and are correlated not independently but jointly with one subjective localization. Consequently, in normal binocular localization there is no independent subjective localization by either eye alone while the other eye is not consciously closed, even though the latter is completely excluded from observing.

OCULAR DOMINANCE AND THE CYCLOPEAN EYE

The so-called cyclopean eye, or the center of visual directions supposedly lying about midway between the two eyes and a little behind them, is purely a hypothetic schema based on the fact that in persons with normal binocular vision both monocularly and binocularly alined objects appear to lie on a line which passes about through this position in the head image. The objects may be alined with one eye alone, the other being open but excluded by a shield, or one may be alined in the fixation axis of one eye and the other in the fixation axis of the other eye, so that each object is seen by only one eye. Binocular alinement is achieved by changing fixation from one object to the other. The understanding of the phenomena of monocular alinement is best approached through analysis first of binocular alinement.

Any one with normal binocular vision can demonstrate to himself the fact that binocular alinement of objects apparently represents a fixed subjective state in the conjugate displacement of the eyes for various degrees of convergence. Thus, when one looks binocularly from a distant to a near object, the two objects will appear to be alined with the observer when there is no subjective change in the conjugate displacement of the eyes on changing the fixation. This phenomenon I shall call the binocular alinement test. From what has been said previously, disjunctive and conjugate movements of the eyes are not consciously represented as such but are represented only in terms of changes in the three-dimensional spatial values of seen objects in relation to the body image, so that absence of subjective change in the conjugate position of the eyes results in changes in convergence being represented consciously as changes in size and distance or depth in a line with the "center" of the observer.

This subjective center is a part of the body image as a whole. As a result of the bilaterally symmetrical arrangements of the parts of

the body, there is a tendency, in the absence of a specifically different intent, to refer subjective, egocentric localization to about the median longitudinal axis of the body image. This axis of reference may be spoken of as the axis of balance for the symmetrically arranged parts, since, at any level, it represents approximately the position of the center of balance. As a whole, it represents also the subjective axis of inertial balance for torsions of the body or its parts. It forms the basis of the subjective median longitudinal plane and thus the origin for the subjective coordinate system of the body image. Objectively, it obviously is not a single straight line but will have various shapes depending on the characteristics of the body.

It might be supposed, with maintenance of the same degree of innervation for conjugate deviations subjectively, that disjunctive movements of the eyes would be objectively symmetrical, that is, the angular rotations of the eyes would be equal but opposite. This supposition, however, ignores certain phenomena on which I believe so-called ocular dominance is based. The usual tests for dominance involve the selection of one eye or the other for monocular alinement or sighting. The explanation for the dominance and its relation to cortical dominance are not obvious from these tests, since one part of each eye is represented sensorially in one half of the brain and the other part of the eye in the other part of the brain and each eye is also subject to motor control from both halves of the brain.

In the binocular alinement test it is common for the objects which are subjectively alined with the axis of balance of the observer to be alined objectively with some point nearer to one eye than to the other. A similar phenomenon has been observed in the alinement of the subject's concealed finger, as when it is under a table or a board, with an object seen binocularly in the distance. The latter test has been used extensively to determine the objective position of the subjective median vertical or longitudinal plane. What the subject actually does is simply to compare the visual localization with the tactile-proprioceptive localization of this plane, for both are subjective. The visual localization coincides with the subjective visual, straight-ahead position which represents physiologically a null point in the innervation for voluntary conjugate movement of the eyes to either side. Its objective position would then depend on factors not under voluntary control and thus chiefly anatomic and physiologic.

Conjugate deviations of the eyes to one side or the other are initiated voluntarily in the portion of the cerebral cortex contralateral to the side of the deviations. It is reasonable, then, to assume a one-sided motor dominance of ocular movements which is analogous to the one-sided dominant development of voluntary motor functions of the rest

of the body contralateral to the dominant portion of the cortex. With dominance of the left side of the cerebral cortex, then, an unconscious hyperactivity of the right conjugate deviators of the eyes should be expected.

If anatomic anomalies of the ocular musculature or orbits are disregarded, the actual position of the subjective visual, straight-ahead position in general would depend on the balance of muscular tonus between the right and left conjugate deviators at the null point of conscious innervation. Hyperfunction of the right deviators would then result in the null point coinciding with more or less deviation to the right and consequently giving a subjective visual, median, longitudinal plane objectively to the right of the actual median plane. Conversely, with right cortical dominance and sinistrality, the left conjugate deviators would be dominant and the subjective visual median longitudinal plane would be objectively to the left of the actual median plane.

The binocular alinement test in conjugate positions other than the straight-ahead position reveals the same effect of motor dominance. Thus, for right or left conjugate positions, binocularly alined objects will be found objectively to be alined more toward the dominant motor side relative to the alinement given by objectively symmetrical convergence. The latter alone would give an alinement toward the side of the actual deviation, as may be shown geometrically. The effect of the dominance, then, is to cause the alinement to be objectively toward the dominant motor side of the alinement given by objectively symmetrical convergence. Any one can test this on himself by noting the positions of the physiologically doubled images of one of the two alined objects relative to the other binocularly fixed object. The alinement by symmetrical convergence would be represented by the binocularly fixed object appearing approximately midway between the two images of the other one, whereas a deviated alinement resulting from unilateral motor dominance would be represented by the former object appearing nearer to that one of the doubled images which belongs to the eye on the side of the motor dominance.

The physiologic effect of the conjugate motor dominance on convergence in the absence of conscious change in the conjugate innervation is thus an objective asymmetry toward the side of the dominance.

My interpretation of so-called ocular dominance according to conjugate motor dominance is that, generally, the directing or dominant eye is the one for which the alinement seems to agree best with the subjective binocular alinement determined by the given conjugate position of the eyes. In this connection it must be borne in mind, from what was said earlier, that even though only one eye is doing the observing while the other is open but excluded, the subjective repre-

sentation of their conjugate position and thus the conscious localization of the seen objects are determined by the correlation between the representations of their respective muscular innervations and a given localization.

A test which I devised several years ago demonstrates, in a manner different from the sighting test, to which side of the head image visual egocentric localization is referred. It depends on the well known fact that, under controlled conditions of observation, such as dark surroundings, a subject with normal, binocular vision who has both eyes open but one excluded by an opaque screen placed in front of it cannot tell which of his eyes is doing the observing. Under these conditions, then, one eye is shielded, and a slender rod is aimed by the examiner either at the inner or the outer canthus of the observing eye. The subject is directed to indicate with his finger at what part of his head the rod appears to point. If it appears to point toward the nonobserving eye, when it actually is aimed at the inner canthus of the observing eye, or if it appears to point to the outer side of the observing eye when aimed at its outer canthus, reference of visually perceived position is assumed to be predominantly on the side of the observing eye, and that eye is termed the "reference eye." If, when the rod is aimed at the inner canthus of the observing eye and the subject indicates that it is pointing toward the outer side of the nonobserving eye, or, when aimed at the outer canthus of the observing eye, it seems to point between the eyes, the nonobserving eye is the reference eye. The results of the test on a number of subjects indicated with few exceptions that the same eye was used for sighting as for subjective reference. Even when the eye used for sighting was not on the side of the motor dominance, the reference eye usually agreed with the sighting eye rather than with the side of motor dominance. These results indicate that the factors determining the sighting eye probably operate also to determine the reference eye.

The customary use of one eye for monocular alinement does not lead directly to the conscious selection of it under the conditions of the sighting or the reference tests but only to the consistent reference of egocentric visual localization to its side of the head image. The basis, then, for the selection of this eye by the sighting test is the better agreement of the apparent subjective visual localizations through it with the localizations determined, respectively, by the conjugate position of the eyes and by tactile-proprioceptive sensations. Thus, in the sighting test as commonly performed, a disk with a hole in it, or a tube, is held by both hands, and a distant object seen binocularly is "sighted" monocularly through it. The position of the distant object is thus given by the conjugate position-localization of the eyes, and the

position of the disk or of the tube is given by the manual tactile-proprioceptive mechanism. The hole or the tube is placed before the eye, which gives thus an apparent egocentric alinement in closest agreement with that given by the aforementioned localizations. If the nonsighting eye is inadvertently used for such tests there is a definite sense of confusion or disagreement in the localizations, and either the manually determined position of the disk or the tube seems wrong or the subject immediately realizes that a particular eye is being used.

Other factors also enter into the choice of the sighting eye, especially the relative visual acuity of the eyes and habit developed in connection with certain tasks. Certain muscular dysfunctions may also play a part. The apparent so-called dominance, determined by the sighting test, is thus simply the result of a selection guided by either a concomitance of these various factors, including the actual conjugate motor dominance, or a predominance of one or more of them.

Of course, mixed types of dominance also would be expected to occur, probably on the basis of a mixed cortical dominance. There is no reason why every person should have complete right or left cortical dominance for all motor activities. Initial training and necessity frequently cause mixed dominance such as is present in the case of left-handed persons who learn to play golf with right-handed clubs or cases of right-handed persons who have a dominance of the left eye in other ways but who aim a rifle by sighting with the right eye.

The phenomena of monocular alinement are based on the same fundamental principles as those of binocular alinement. The objects, which are observed monocularly, whether one is in the fixation axis of one eye and the other in that of the other eye or both are in the same fixation axis, have their egocentric subjective localizations given according to the four correlations previously described. In monocular alinement both conjugate and disjunctive innervations are held in their existing subjective state. The only clue, then, to the relative egocentric localizations of the objects is in their size-to-distance relationship, the nearer one appearing the larger and being correctly interpreted as nearer, or if they are both on the same fixation axis the additional factor of the nearer one obscuring all or part of the distant one confirms this judgment. As was brought out in discussing binocular alinement, changes in convergence in the absence of subjective change in the conjugate position of the eyes are represented as changes in size and depth distance in a line with the subjective center, or axis of balance, of the observer, objective deviations occurring toward the side of the conjugate motor dominance irrespective of asymmetry of convergence. Thus, this line represents the locus of subjective egocentric localizations with which various convergent positions of the eyes are correlated when there

is no subjective change in their conjugate position. Consequently, in monocular alinement, the subjective egocentric (depth) localizations determined by the size-to-distance relationship are correlated with their respective convergence innervations and seen in the locus of positions determined by these, thus apparently in a line with the subjective axis of balance of the observer.

According to these interpretations, it is difficult to see any basis for assuming a primary center of visual directions as implied by Hering's rule. If one were to limit oneself to the use of directions alone without three-dimensional localization in interpreting the phenomena of alinement, one would have to assume a primary center of reference for these directions. Thus, if one accepts identity of directions as the criterion for retinal correspondence, then, as Helmholtz [3] has stated, one must assign a center of reference for the directions. But if one considers the fact that the monocularly observed objects are individually localized subjectively in three dimensions, the position in the head image of this so-called cyclopean center of directions, as indicated by the phenomena of alinement, is seen to be simply the physiologic consequence of the fundamental subjective correlations which determine subjective egocentric localization. It is thus not a fundamental subjective center of reference for visual direction but is merely the result of the visual interpretation of the subjective axis of balance of the body image.

SUMMARY

Subjective egocentric visual localization is the determination of the position of seen objects in relation to the body image of the observer. It is accomplished by means of four fundamental correlations of innate origin: (1) the correlation of the fixation intent with simultaneous, binocular foveal fixation; (2) the correlation of the positions of the individual foveal (fixation) visual axes with the innervational states of the respective extra-ocular musculatures, these positions being represented subjectively in terms of the innervational states; (3) the simultaneous, joint correlation of the positions of the individual foveal (fixation) visual axes, as given in these terms, with a single subjective egocentric localization in three dimensions, and (4) the correlation between these innervational representations and the apparent size of objects, which indirectly furnishes the third dimension, or depth, through size-to-distance relationships.

Subjective egocentric visual localization is always three-dimensional, whether the observation is made monocularly or binocularly and whether

3. Helmholtz, H. L. F.: Helmholtz's Treatise on Physiological Optics, translated from the German edition and edited by James P. C. Southall, Rochester, N. Y., The Optical Society of America, 1925, vol. 3, p. 243.

or not there may be agreement with objective localization or accuracy of expression of the localization in objective terms.

Retinal correspondence denotes a functional relationship between individual retinal points in the two eyes such that percepts originating from them have an innately fixed mutual correlation with a single subjective egocentric localization in three dimensions. Definition of correspondence in terms of direction is incorrect as well as inadequate.

The phenomena of binocular and monocular egocentric alinement are based on the four fundamental correlations involved in subjective egocentric localization with the additional factor of true unilateral motor dominance. The latter exerts its effect through hyperactivity of conjugate deviation toward the side of the motor dominance, therefore contralaterally to the dominant half of the cerebrum. The alinement is the result of the maintenance of conjugate deviation in the existing subjective state and is determined by the locus of subjective egocentric localizations which are correlated through the size-to-distance relationship with the various convergence innervational states for this subjective state of conjugate deviation. Because the localizations are all in relation to the body image and because of the approximate symmetry of the parts of the latter about the subjective axis of balance to which localizations in general are referred, this locus is directed toward the axis of balance also. The point toward which it actually appears to be directed represents, therefore, not a primary center of visual directions (the so-called cyclopean eye) but merely the interpretation of the subjective axis of balance in terms of subjective egocentric visual localization. Because of the effect of unilateral motor dominance on conjugate deviation, this point is displaced toward the side of the motor dominance.

A new interpretation is given so-called ocular dominance, based on the effect of unilateral motor dominance on conjugate motor deviation. The dominant eye as selected by the sighting test is the one for which the alinement thus determined usually agrees best with the subjective alinement based on the given conjugate position of the eyes. Thus, if the conjugate position of the eyes has suffered a deviation to one side as the result of motor dominance on that side, sighting with the homolateral eye will give an alinement which agrees better with the subjective egocentric positions of the target and the sight than that given by sighting with the other eye. Habit or training and the relative visual capacities of the two eyes are also factors in determining the sighting eye.

Clinical Notes

A CASE OF LYMPHOCYTIC MENINGITIS (?) WITH OCULAR FINDINGS

GERALD D. SPERO, M.D., DETROIT

Benign lymphocytic meningitis seems to be on the increase. The disease is peculiar in that it is characterized by a severity of symptoms that is surprising, in view of the fact that recovery is prompt and complete. The condition occurs chiefly in younger persons.

REPORT OF CASE

History.—S. A., an Italian youth aged 15, was referred to me on Aug. 18, 1936. His chief complaint was photophobia. With the exception of scarlet fever a number of years previously, he had always enjoyed good health. About one week prior to my seeing him he began to complain of extreme photophobia. This was accompanied by headaches, with no particular localization. These symptoms were constant. Dark glasses did not help. Salicylates helped only a little.

Examination.—At this time a general examination showed nothing of importance. The ocular examination revealed the following:

Vision in each eye was normal. Refraction was carried out with the employment of homatropine, and with the eyes under the influence of drops the patient accepted a + 0.75 D. sph. combined with a + 0.25 D. cyl., ax. 90 for each eye. With this correction, vision in each eye became 20/20 + 4. The extra-ocular movements were normal. The pupillary reflexes, media and tension were normal. Tests of the ocular muscles showed nothing of importance. The right disk was slightly hazy nasally, but no swelling was present. However, the entire left disk was swollen, to the extent of from 0.5 to 1 D. The swelling was confined entirely to the disk. The veins around the disk were somewhat engorged, the arteries being normal. The picture was suggestive of early choked disk. The field for color and that for form were normal in each eye. No central scotoma was present in either eye. The record of the ocular findings was turned over to the attending physician, and he in turn called in a neurosurgeon, Dr. S., for consultation. The latter advised hospitalization.

Subsequent Findings and Course.—The subsequent findings and the course in the hospital were as follows: Shortly after admission the temperature rose to 102 F., and the lymphatics (axillary, inguinal and cervical) became slightly swollen. During the first few days the patient vomited several times (the vomiting being projectile in type). In addition to the photophobia and headaches, dizziness was noted. The patient also complained of mild pains in the muscles of the calf. The pulse rate on admission was 66. The respiratory rate was 18.

Shortly after admission a lumbar puncture was done, and the fluid was found to be under some pressure and clear. No organisms could be demonstrated. Nine hundred cells were present. Of these, 72 per cent were lymphocytes and 28 per cent polymorphonuclears. The colloidal gold chloride test showed nothing of importance. The Kahn reaction was slightly positive. The test for chlorides

showed 726 mg. of sodium chloride per hundred cubic centimeters. The globulin content was slightly increased. The sugar content was found to be 0.073 per cent.

Examination of the blood on admission showed the hemoglobin to be 95 per cent and the red cell count to be 4,720,000. The white cell count was 14,900. The differential count was as follows: stab cells, 8 per cent; segmented cells, 70 per cent; lymphocytes, 20 per cent; large monocytes, 2 per cent. The urine had a specific gravity varying between 1.012 and 1.008 and contained no albumin or sugar. On admission a trace of acetone was found, but subsequently acetone was absent.

On October 23 the pressure of the spinal fluid was still somewhat elevated, but the number of cells had dropped to 130, and of these 92 per cent were lymphocytes and 8 per cent polymorphonuclears. The complaints had started to lessen in severity. On October 24 the spinal fluid was clear and under no pressure. On October 26 the patient was free from all complaints, and since the spinal fluid was normal he was sent home. Several weeks after his discharge from the hospital the patient felt fine, had gained some weight and returned to work. It seems that his complaints started to disappear with the first lumbar puncture and were gone the day after the third one. When he was last seen (several weeks after discharge from the hospital) the disks had become completely normal.

COMMENT

Differential Diagnosis.—Encephalitis: The presence of choked disk and the absence of elevation of the level of sugar in the cerebrospinal fluid speak against encephalitis in the case reported. In some cases, though, differential diagnosis between encephalitis and lymphocytic meningitis may be difficult.

Poliomyelitis: This condition may be present, and probably frequently is, without paralysis, causing signs of general infection with or without localizing signs in the central nervous system. In the case reported here, however, the cell count of the spinal fluid at the original examination was much higher than that found in infantile paralysis.

Tuberculous Meningitis: Tuberculous meningitis is easily ruled out in the case reported in this paper by the fact that the patient recovered, and it was not seriously considered in the acute stage because of the normal sugar content of the spinal fluid, the absence of the tubercle bacillus in the spinal fluid and lack of evidence of any systemic tuberculosis.

Other Diseases.—In epidemic meningitis, as well as in all bacterial forms of meningitis, examination shows the causative organisms in great profusion and without difficulty. Further, these conditions are usually characterized by higher cell counts and the presence of a much larger proportion of polymorphonuclear leukocytes than was found in the case just reported.

Syphilis of the central nervous system presents a manifold and often bizarre clinical picture, depending on the extent of involvement and the areas affected. In syphilis sufficiently active to produce the symptoms noted in the case reported one would certainly expect a strongly positive serologic reaction of the spinal fluid and a

more atypical colloidal gold curve than was obtained, even though the serologic reaction of the blood might be negative. Further, the age of the patient and his antecedent history speak strongly against this diagnosis.

A clinical syndrome known as meningitis aseptica, which is an inflammatory process of the meninges due to nonbacterial causes, has been described in the literature. It has followed injections of certain irritating medications and serums, as well as saline irrigations of the spinal canal. None of these factors, however, was responsible in the present case.

CONCLUSIONS

For want of a better term I am calling the condition in the case reported in this paper acute lymphocytic meningitis. Others call it aseptic meningitis. In any event, the disease is mild, and a good prognosis can be given. Many of the patients show no ocular findings. In my case a choked disk was present. Other authors have reported a paretic squint, which usually clears up. Clinically, the outstanding complaint is photophobia. Headaches and dizziness may also be present. Early in the disease an excessive number of polymorphonuclears may be present, and lymphocytosis may exist later. The total protein content of the spinal fluid is increased, and globulin is invariably present. There is only a slight reduction in the amount of dextrose and chlorides in the spinal fluid—an important feature distinguishing this condition from tuberculous meningitis, in which a definite and marked diminution of the amount of chlorides and dextrose is always present. The average duration of the disease is seven days. The temperature falls by lysis, and, as a rule, complete recovery takes place in two or three weeks. This condition is now generally regarded to be due to a distinct virus recently recovered (in 1934), and thus the term aseptic meningitis would, scientifically, be a misnomer. Examination of the spinal fluid is necessary to establish a diagnosis. The treatment is largely symptomatic. Repeated spinal punctures relieve the photophobia and headaches. The disease affects mostly young persons (those from 20 to 28 years of age), and, as a rule, there is no history of an infection of the ear or the nasal sinuses. This clinical syndrome should be borne in mind. As I look over past histories it seems to me that some of the conditions previously reported might have belonged to this group.

A NEW CALIPER

JOHN GREEN, M.D., ST. LOUIS

Accurate measurement from the line of insertion of any rectus muscle to the point on the sclera where it is desired to recede or advance that muscle is necessary. For this purpose a number of calipers have been designed. I have not been satisfied with any so far placed on

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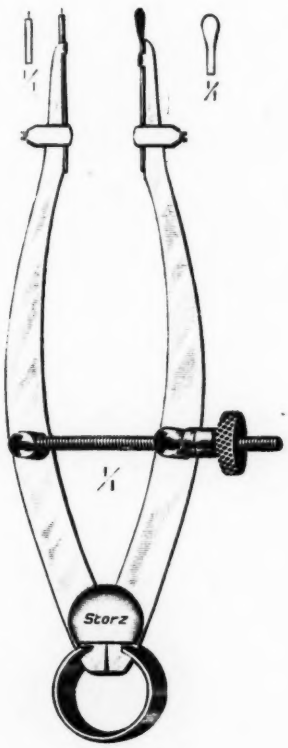
the market. One type, designed on the principle of a slide rule, measures accurately the desired distance, but the projecting bars are dull and do not give an opportunity to indicate by a delicate scratch mark the position on the sclera for the insertion of the sutures. Another type is a small caliper with dull points neither of which serves the particular purpose desired.

I have had an instrument maker construct a caliper one shank of which carries a thin shaft of steel which terminates in a very fine sharp point. This point cannot penetrate the sclera, as it is guarded by the



The new caliper.

shaft. The other arm of the caliper carries a small round-ended knife, which can be removed for sharpening, if necessary.

In use the point and the knife are separated to the desired distance (e. g., 5 mm.) by means of the adjusting screw.

The sharp point penetrates the upper layers of the sclera at either the upper or the lower border of the tendon at its insertion, and a tiny scratch mark is made on the sclera with the round-ended knife. This scratch mark is fine but sufficiently distinct so that when the sclera is freed of blood it is possible to place a suture precisely.

The caliper has been found of great use in Curdy's recession operation and also in Curdy's advancement operation.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

SOME PROBLEMS AND PROCEDURES IN REFRACTION

AVERY DeH. PRANGEN, M.D.

ROCHESTER, MINN.

Refraction calls for a technic of painstaking mathematical accuracy combined with clinical judgment, that valuable art the anlage of which is a physiologic point of view—the ability to visualize the human machine as a whole, each part in relation to the other, and to see the mechanism at work, sensing dysfunction, incoordination, conflicts, defects and organic troubles in their true relation and importance to the patient as a living mechanism. In other words, the ophthalmologist should not confine his interest, knowledge and attention to the narrow limits of his special field so that he goes off on a tangent and fails to comprehend the theme of the complete clinical picture. Diagnosis and treatment in a specialty must fit in accurately with the numerous and varied factors making up the many jagged parts of the complete clinical picture. Failure to take this into account is perhaps the outstanding criticism of specialization. After all, the ophthalmologist is the physician practicing ophthalmology.

With the foregoing considerations in mind, it may be stated that refraction has for its major purpose the alleviation of symptoms of ocular distress by the proper fitting of glasses. Local medicinal treatment, orthoptic exercises and surgical measures are necessary and are valuable adjuncts when intelligently used. In the prescribing of glasses, science and the art of practicality must be deftly mixed, it being ever remembered that the acid test is the patient's approval. The prescribed glasses must give clear, ready vision and enable the patient to carry on his work and play with comfort and with a minimum of fatigue. To accomplish this constitutes the fine art of refraction.

In the practice of refraction much trouble and dissatisfaction can be avoided by keeping in mind certain pertinent relationships existing between the ophthalmologic, general medical and neurologic aspects of the case. The ocular symptoms of which a patient complains may

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This material is from a series of postgraduate lectures given at various times at Rochester, N. Y.; Cleveland; Ann Arbor, Mich.; Madison, Wis., and New York.

be but reflex or mirrored disturbances arising from a disordered mental or physical state. The eyes, by virtue of their origin from, and their intimate connections with, the brain and the central nervous system, are prone to reflect disease and functional disorders of these structures. Neurasthenia, psychoneurosis, traumatic neurosis, hysteria, mental inadequacy and similar disorders usually cause symptoms of ocular distress which glasses or treatment of the eyes will not cure. The prescribing of glasses for the unfortunate persons with such a disorder brings nothing but trouble for the physician. Increased intracranial pressure from tumors and abscesses of the brain causes decrease in vision, and glasses are not the answer; atrophy of the optic nerve and optic neuritis affect vision likewise, and the cause must be looked for elsewhere than in the eyes. The ophthalmoscope and the perimeter must be constantly employed to avoid the pitfall of attributing all disturbances of vision to an ocular cause. Toxic amblyopia resulting from tobacco, alcohol or some other poison or due to syphilis may be interfering with vision, and the ophthalmologist must be quick to note the presence of such a factor. Retinitis from arteriosclerosis, hypertension, pregnancy, anemia, diabetes, renal disease or some other condition may be causing visual difficulty. The asthenic, run-down and generally ill patient naturally has no ocular comfort. Those recovering from recent general surgical and medical treatment often complain of inability to use their eyes. All these and many other neurologic and medical disorders secondarily affect the eyes, and the alert ophthalmologist hesitates to prescribe glasses until such time as the patient has been restored to a reasonable degree of health.

Age and senility also bring about a decrease in ocular capacity. One should change glasses for aged persons with caution, as dissatisfaction with new glasses is the rule. It is not possible to enable these patients to use their eyes comfortably and as much as they wish with any glasses. Persons working at high tension, which creates nervous exhaustion, have much ocular fatigue, and glasses will not correct this. The local, social, economic and familial status must also be taken into consideration. Thus, when a patient comes in for refraction the ophthalmologist must consider all these various factors and attempt to evaluate the condition of the patient and consider whether it is feasible to undertake to give relief by ocular treatment or glasses. By such a differentiation much unpleasantness can be saved the ophthalmologist and serious trouble and dissatisfaction can be saved the patient.

A problem which has interested me for a long time is the basic difference clinically between various types of eyes. There is a sharp contrast in the visual capacity and reactions of the emmetropic, the myopic, the hyperopic and the astigmatic eye. One cannot expect the

same performance from all eyes. They differ from one another in their reactions and capacity, just as one person varies from another. It is important and useful to keep this in mind when evaluating ocular symptoms and attempting to prescribe for them. The clinically ideal eye is perhaps the eye which does not vary too far on either side of emmetropia and has not too much astigmatism—in other words, an eye near the normal in the curve for frequency distribution of refractive errors. Such an eye, whether it is slightly hyperopic or myopic, as a rule carries on its functions well.

The more eyes deviate, however, from the normal toward the higher degrees of ametropia and the more they are affected by marked astigmatism, the more difficult becomes their task of carrying on. Glasses, as is known, do not make eyes normal, nor do glasses enable eyes with marked errors of refraction to perform with the same ease as those eyes near normal. It is therefore natural to expect that markedly ametropic eyes, though well corrected, should give much more discomfort and have less capacity for ease of function. The normal eye is an effortless mechanism, and the possessor of such eyes is to be envied. The markedly ametropic eye, on the other hand, has to be whipped and spurred to make it accomplish its tasks. When, in addition, anisometropia and antimetropia exist, differences in the amount and the kind of error in the two eyes, the difficulty of proper ocular performance becomes even more exaggerated. In these markedly anisometropic and antimetropic eyes there exists an essential and intrinsic irritability which even the best of corrections usually fails to subdue completely. Persons with these eyes often get into a bad state ocularly and mentally through lack of understanding of the true nature of their ocular mechanism by themselves and by their ophthalmologist. Not obtaining relief, they relentlessly pursue comfort and go from physician to physician. Having tried numerous corrections and much varied advice and continuing to be uncomfortable, they naturally may assume that there is something radically wrong with their eyes. They come to feel that they are faced with eventual ocular disaster.

Although this seems a logical conclusion to them, one knows that it is not true. It is a false conclusion drawn through misinformation and lack of understanding. It makes a fine basis, however, for a profound ocular neurosis. If the ophthalmologist understands the nature of such eyes and their essential irritability, it is relatively easy to put the patient at ease. Eyes like these are a nuisance, not a menace. They are an inherited handicap which must be overcome by perseverance and effort. Even with the best correction possible, they will never be any too comfortable, but no harm is done to the eyes in making them work. The fatigue, ocular and nervous, which results from this effort is relatively harmless when understood and philosophically endured.

It is the price to be paid for the things which one's ambition wishes to accomplish. The eyes will not be harmed in any way by such usage. Viewed in the light of this reasoning, when understood by the intelligent patient, the problem of the irritable anisometropic, astigmatic eyes is not difficult. In my experience, the reaction of the distressed persons with such eyes to this explanation is both grateful and satisfactory. Yet they constitute a rather large, troublesome and difficult group of patients in the average practice.

In connection with anisometropia and antimetropia, attention must be called to the work being done on aniseikonia, or difference in size of the images in the two eyes. This is an important work and should be watched. It is still in the experimental stage and has not yet been reduced to clinical application for those ophthalmologists engaged in office practice. Without doubt, in the near future the studies of this subject will have been thoroughly evaluated and put on a clinical basis.

There is also a definite and basic difference between hyperopic and myopic eyes. I formerly thought that the designations far sighted and near sighted were misnomers. I believe now, however, that they are good names. There is no doubt that myopic, or near-sighted, eyes are designed to do near work with ease and efficiency, whereas distance vision is more difficult for them. With hyperopic, or far-sighted, eyes the reverse is true. It is true that both near-sighted and far-sighted eyes can be made to do all kinds of work, but each excels in its own field. If they are called on to do work opposite from that for which they were designed, naturally more difficulty will be encountered.

The ability of the eyes to focus properly both for distance vision and for near vision is always a determining factor in ocular comfort. The status of the accommodative power and its relation to convergence constitute one of the most important problems in refraction. A knowledge of the state of the accommodation of each patient is absolutely necessary in the proper treatment of the condition. It determines not only the needs of the patient in regard to the strength of lens but by what methods he should be examined and what cytoplegic, if any, and how much should be used. One should measure the accommodative power in every case, the data thus obtained being indispensable in the evaluation of the eyes in question. As one does this one comes to rely more and more on this useful information. The normal accommodative values have been worked out carefully by Jackson [1] and Duane [2] and are

1. Jackson, Edward: The Amplitude of Accommodation at Different Periods of Life, and Its Relation to Eye-Strain, California State J. Med. **5**:163-166 (July) 1907.

2. Duane, Alexander: Fuchs' Text-Book of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, 1924, pp. 154 and 178-211.

available to use as a guide. To avoid repetition I refer the reader to two previous publications [3] which deal with this subject.

In discussing abnormal accommodative states, the following classification, modified after the arrangement by Howe,[4] may be useful:

1. Normal, near point of accommodation, average for age
2. Subnormal, near point of accommodation, below average for age
   (*a*) Paresis
      (1) Static or premature presbyopia
      (2) Dynamic or accommodative insufficiency
   (*b*) Paralysis
3. Spasm, relative excess of—near point of accommodation near normal
   (*a*) True spasm } associated with ametropia
   (*b*) Spasticity }
4. Abnormally powerful or sustained—near point of accommodation close
   (*a*) Actual excessive, with ametropia
   (*b*) Actual excessive, with emmetropia

Of the conditions listed in this classification, I shall discuss briefly dynamic insufficiency, spasm and abnormally powerful accommodation. Certain instances of these three forms of accommodative disturbances are most interesting. Although such disorders are not common, when they do occur they cause marked difficulty for the patient and are likely to be overlooked by the ophthalmologist.

Subnormal accommodation manifests itself by inability to do near work or to sustain it in younger persons, those aged from 15 to 35 years of age. As persons of this age are supposed to have ample accommodative power, the condition will not be noted unless the accommodation is measured as a routine. The symptoms are those of accommodative asthenopia, and the accommodation will be found to be consistently below the average normal for the age. In these cases there seems to be a lack of that reserve potential accommodative power that usually is held in readiness to make near vision effective. Analysis of cases observed at the clinic showed that the condition was due to asthenia, debilitating conditions and focal infection, particularly dental sepsis. In some cases it seemed that the person naturally possessed a low accommodative power. Treatment was to relieve, if possible, the source of the trouble, after which the accommodation returned to normal in some cases; in other cases, however, the patient had to be given more plus lens for near work. For a fuller discussion of these cases I refer the reader to a previous publication.[3b]

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3. Prangen, A. DeH.: (*a*) What Constitutes Satisfactory Cycloplegia? Am. J. Ophth. **14**:665-671 (July) 1931; (*b*) Subnormal Accommodation, Arch. Ophth. **6**:906-918 (Dec.) 1931.

4. Howe, Lucien: The Muscles of the Eye, New York, G. P. Putnam's Sons, 1908, vol. 2, p. 26.

Spasm of accommodation is, I believe, closely related to dynamic subnormalcy. In a previous paper[5] I reported thirty cases of this condition. Like subnormal accommodation, it occurs in younger persons, those aged from 15 to 25 years. Characteristic of the difficulty is the apparent rejection by these eyes at the postcycloplegic test of proper correction of their hyperopia or a tendency to exaggerate their true correction for myopia as found with cycloplegia. At the postcycloplegic test or during a manifest refraction they will accept minus lenses when their error calls for plus lenses. The name spasm applied in these cases is misleading, as it perhaps implies excessive power; in reality, the condition seems to be one of irritability due to asthenia. These eyes are extremely uncomfortable unless relaxed by proper correcting lenses instead of a pseudomyopic correction. The symptoms are blurred distance vision, headaches, ocular discomfort and inability to use the eyes without fatigue. The accommodative near point in most cases is about normal for the age of the patient, although in a few cases it is intermittently too close. The primary cause of the phenomenon appears to be long-continued strain from attempting to adjust the ciliary mechanism to insufficient or improper correction, and this underlying cause is reenforced by secondary factors, such as functional disorders and focal infection. It is to be noted that the disturbing factor of anisometropia is almost always present. The nature of the trouble seems to be a cramp due to fatigue of an overworked and usually asthenic ciliary mechanism. In some respects a weak ciliary muscle and a spastic ciliary muscle are much alike. When subjected to undue fatigue the weak muscle gives up, whereas the spastic muscle continues to act in a spasmodic and inefficient manner. In either instance there is evidence of accommodative asthenia. The treatment of spasm is a proper correcting lens and a balance of the anisometropia. These eyes require a more accurate and balanced correction. At first the patient appears to refuse this correction, but usually within a few days or with the help of a little more homatropine he accepts the correction with gratitude. As a matter of fact, since I realized that the condition was one of irritability due to asthenia I have seldom used any more cycloplegic to relax the eyes to the new correction. The eyes adjust by themselves quickly. Of course one should bear in mind the age of the patient and not try to blur distance vision by too much spherical correction, as patients with this disorder are usually young. Also, anisometropic eyes are essentially irritable in spite of any correction. The patient should understand this and should not rush to get a new correction every time he has a relapse of symptoms, which occurs

5. Prangen, A. DeH.: Spasm of Accommodation, with Report of Thirty Cases, Tr. Sect. Ophth., A. M. A., 1922, pp. 283-292.

frequently. With stress, strain and fatigue the spasm is prone to recur. It will soon disappear again, however, with the continued wearing of the proper glasses and a return of physical and mental balance.

One occasionally observes instances of abnormally powerful or sustained accommodation which is the opposite of the conditions just described. It may occur in association with either ametropia or emmetropia. When seen in association with emmetropia, the condition is one of late presbyopia, the patients being 50 and even 60 years of age and not seeming to need reading glasses. One observes such instances every now and then. In association with ametropia one sees powerful accommodative mechanisms which seem to be able easily to carry marked hyperopic errors even at the ages of 38 to 43 years without the use of correcting lenses or possibly with the use of only low plus lenses for near work. Aside from interest in the unusual occurrence of such cases, the clinical point is the folly of trying to force glasses on these fortunate persons or to give them more than a mere minimum of correction for their needs. Their accommodation is such that they need little or no assistance.

Balance and imbalance of the extra-ocular muscles have always been a diffuse and difficult ophthalmologic problem. The diagnosis and treatment often seem obscure. I think it is possible to make this problem more simple by refining still further the usual conception of diagnosis. Too often diagnosis of the muscular condition stops with the statement that the patient has exophoria or esophoria, in distance vision or in near vision. No further effort is made to coordinate the relation of the phoria in distance vision to that found in near vision and the relation of the phoria to the powers of lateral and vertical duction.

There appear to be four steps in the completion of a refined diagnosis of a muscular condition: (1) the finding of a phoria (esophoria, exophoria or hyperphoria), (2) the determination of the degree of deviation for distance vision and for near vision and a comparison of these figures, (3) a study of the duction powers and (4) a study of the behavior of the individual muscles in their primary field of action. With these data at hand one is in the position to make an exact clinical diagnosis. The figures recorded are not of so much importance, I feel, as is a careful comparison of the relationships of the aforementioned data to one another. By putting together the information found from these four procedures the clinical diagnosis becomes apparent. All anomalies must be disturbances of convergence or divergence of the visual axes, either lateral or vertical. The table shows my arrangement of the points considered in this method of diagnosis. Following this line of reasoning, one can easily determine just what type of anomaly one has to deal with. It is sometimes true that deviations are the

same for distance vision and near vision. This is evidence of a compensated condition of long standing, which nature has rendered less troublesome by causing it to become continuous or constant. A little further study along the lines already mentioned will enable one eventually to figure out the true diagnosis. After the diagnosis has been made, the treatment, whether optical, medical or surgical, becomes much more clearly indicated. The type of treatment is naturally a matter of training, experience and personal preference. It is constantly changing and improving. I firmly believe, however, that a more refined and accurate differential diagnosis greatly simplifies the proper handling of imbalance.

An important point in the handling of imbalance is the constant association of anisometropia with conditions of this type. It may well be possible that uncorrected and improperly corrected errors of refraction, particularly when anisometropic, are the source of irritation and fatigue, which bring about the imbalance. If so, proper correction of

Excess and Insufficiency Under Various Conditions

Deviation Greater In	
Distance Vision	Near Vision
1. Exophoria a. Excess of divergence	1. Exophoria a. Insufficiency of convergence
2. Esophoria a. Insufficiency of divergence	2. Esophoria a. Excess of convergence

refractive errors is the first and most important step to be taken in the treatment of apparent muscular imbalance. Many times this is all that is necessary. If symptoms persist after a thorough trial of proper glasses, one must of necessity consider other measures, such as orthoptic exercise and surgical intervention. In a previous paper [6] I discussed the relationship between the differential diagnosis and the selection of operative procedures. I believe that a careful and refined differentiation of cases makes possible the selection of that combination of surgical intervention best fitted for each individual case. By this process of differentiation and selection it seems possible not only to obtain better cosmetic results but to arrive at the ideal of more normally functioning eyes.

This paper may be divided into two sections. In the preceding part some clinical aspects of refraction have been considered. The following observations pertain more to problems of technic and prescription.

6. Prangen, A. DeH.: Surgery of the Rectus Muscles of the Eye: Selection of Operative Procedures by Differential Diagnosis, Tr. Am. Ophth. Soc. **32**:273-283, 1934.

PROBLEMS OF TECHNIC AND PRESCRIPTION

The patient who returns dissatisfied presents a difficult problem because he is likely to be handled improperly. The fact that he returns complaining or still uncomfortable implies that perhaps a mistake or an error in judgment has been made. It is natural for the examiner to resent this implication, and it is difficult to receive such a patient properly. The patient who returns dissatisfied is a challenge to the ability of the ophthalmologist. That he bothers to return is a compliment, in that this implies he still believes the ophthalmologist can help him and he is offering the ophthalmologist another chance if he will take it. Swallowing his pride, the ophthalmologist should receive him with good grace and disarm him by a pleasant willingness to try and set matters right. The ophthalmologist really should be glad that he returns, for often something needs adjustment. There may have been an error in the manufacture or adjustment of his glasses, or something in the prescription or findings may not check. After the ophthalmologist has received the patient decently it is relatively easy to correct anything found amiss, whereas an uncompromising attitude would have forced the patient to go elsewhere. These cases are not common, so that when they do occur one can well afford to recheck the findings completely in search of some unusual factor, error or overlooked detail. If after a careful recheck everything seems all right, the patient is usually willing to persist with the treatment or correction, for he has been pleasantly and tactfully received and thoroughly examined. The only remedy for an error or oversight is to recognize and correct it.

The indications for the use of a cycloplegic and its dose and sufficiency are of interest to all doing ophthalmologic refraction. A cycloplegic is used to relax the muscles of accommodation when accommodation is of an amount to interfere with the finding of the latent refractive error. When from age or illness the accommodative power has been reduced to 4 D. or less, cycloplegia is not, as a rule, employed, for such an accommodative power does not interfere with the finding of the total refractive error. Persons with such accommodative power usually have presbyopia and are more than 44 years of age. The dose and sufficiency of the cycloplegic seem to be indicated by these facts. In younger patients with more active accommodation, I believe that degree of cycloplegia is needed which will reduce the accommodative power to the level of presbyopia during the process of refracting the eyes. During the course of cycloplegia, as the accommodative level falls to less than 3 D. and approaches 2 D., the total refraction becomes easily manifested. In a large series of cases studied I found that a residual accommodation of 1.5, 1.7, 2 and 2.2 D. was satisfactory.

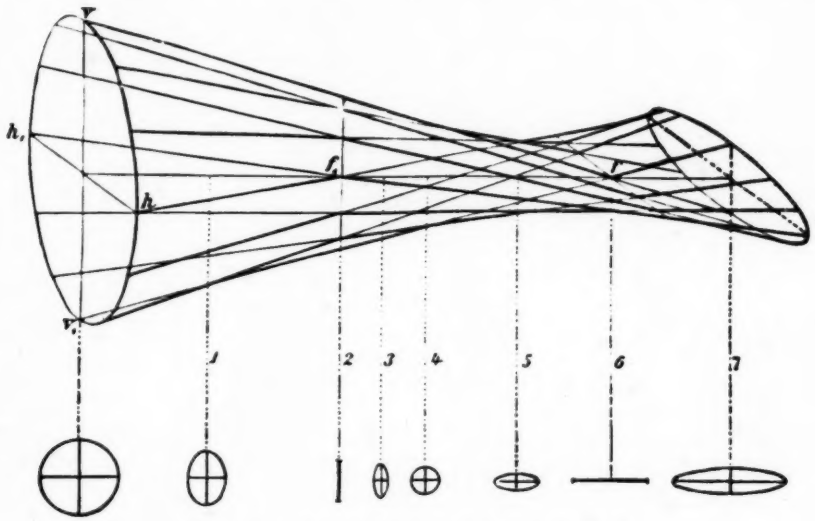
No additional latent error was found below these figures. As the accommodative power rose toward 3 and 4 D. the findings were uncertain and inaccurate. One can easily measure the residual accommodative power at the conclusion of a refraction in which cycloplegia was employed by adding a + 3.00 D. lens to the findings and measuring the accommodative range, the difference between the far point and the near point, in centimeters. This difference in centimeters, divided into 100, equals the range or remaining accommodative power, in diopters. This procedure enables one to determine the degree of cycloplegia obtained. One soon learns also, knowing the accommodative power of the individual patient, what dose of cycloplegic is needed to reduce the accommodative power to a satisfactory level in order to find the total refractive error without hindrance by the accommodation. The usage, dose and sufficiency of the cycloplegic, then, become a matter of measurement and adaptation to the individual patient rather than a matter of purposeless routine.

The therapeutic use of cycloplegia should also be kept in mind. Ophthalmologists often use it to allay ciliary irritability, spasm and fatigue. Eyes are put at rest and induced to accept the desired correction and to overcome bad ciliary habits resulting from insufficient or improper correction. A cycloplegic often is used in this way for patients who are in the late forties and early fifties, as well as for younger patients. I think that ophthalmologists are likely to forget these facts when confronted by persistently uncomfortable eyes.

The conception of astigmatism and its treatment is aided greatly by keeping in mind a picture of Sturm's astigmatic interval (shown in the figure). This interval is the space between the two principal focal points of an astigmatic lens system. The series of distorted images, occurring at different points in the interval, are representative of the images seen by an astigmatic eye when viewing an object. The clinical problem in astigmatism is to replace these images with the corrected image by obliterating the interval by means of a correcting lens, in other words, to bring the two principal focal points together. This would be a full correction of the astigmatism, an ideal. It is often necessary to give the eye a compromise image or partial correction for the astigmatism by partially obliterating the interval or bringing the focal points closer together. In initial attempts to correct higher degrees of astigmatism, the eye, being used to a distorted image or wide interval, may take more kindly to a gradual adjustment of the interval by partial or compromise corrections. There seems to be no logical reason why all eyes should be required to adjust to immediate full correction. The patient and the ophthalmologist often will be much happier when astigmatism is corrected gradually over a period of time. The tendency of American ophthalmologists, myself included,

is to insist as a routine on full correction for astigmatism. Among young persons and those already wearing partial corrections this works well. Among older persons who are unaccustomed to correction for astigmatism there are, I feel, many whose astigmatism should be corrected partially and gradually.

I know of no rule which tells how much to deduct when the full astigmatic error is known and one wishes to correct it partially. This one must decide for oneself. There is, however, a rule of spherical and spherocylindric equivalents of formulas for the correction of astigmatism which is a great help. This was called to my attention by Mr. J. C. Copeland,[7] of the Riggs Optical Company. The spherical equivalent of any formula for the correction of astigmatism is equal to the



Sturm's astigmatic interval (after Fuchs).

value of the spherical lens plus half the value of the cylindric lens, added algebraically. There exists, therefore, a series of equivalent formulas for any given formula for the correction of astigmatism. If the cylinder is reduced by any given amount, half of this should be added algebraically to the existing sphere. By trial of a series of cylindric reductions in their equivalent formulas one can be found which gives the patient vision comparable to the full correction. This is, of course, another variation of Sturm's interval. The patient is allowed to select a compromise formula or image which obliterates the interval partially instead of fully. I have found it better to use these equivalent formulas than merely to reduce the cylinder and make no change in the

7. Copeland, J. C.: A Study in the Application of Cylindric Corrections, Optometric Weekly, April 5, 1928.

sphere. It is also useful to form the habit of thinking in terms of equivalents when one is comparing and evaluating lenses and formulas.

Often lenses worn by a patient when compared to one's findings appear divergent. Closer scrutiny, however, may disclose that they are equivalents, and the one may be as useful to the patient as the other. As an example in the consideration of equivalent formulas, the full acceptance with a manifest test or a postcycloplegic test could be: for the right eye a —2.00 D. sph. + 5.00 D. cyl., ax. 90. The patient may be 39 years old and may never have worn glasses. The equivalent formulas are:

(1)	—2.00 D. sph.	+5.00 D. cyl., ax. 90	= equivalent +50 D. sph.	Equivalent = sphere + ½ cylinder (added algebraically)
(2)	—1.75 D. sph.	+4.50 D. cyl., ax. 90	= equivalent +50 D. sph.	
(3)	—1.50 D. sph.	+4.00 D. cyl., ax. 90	= equivalent +50 D. sph.	
(4)	—1.25 D. sph.	+3.50 D. cyl., ax. 90	= equivalent +50 D. sph.	
(5)	—1.00 D. sph.	+3.00 D. cyl., ax. 90	= equivalent +50 D. sph.	
(6)	—0.75 D. sph.	+2.50 D. cyl., ax. 90	= equivalent +50 D. sph.	
(7)	—0.50 D. sph.	+2.00 D. cyl., ax. 90	= equivalent +50 D. sph.	
(8)	—0.25 D. sph.	+1.50 D. cyl., ax. 90	= equivalent +50 D. sph.	
(9)	0 D. sph.	+1.00 D. cyl., ax. 90	= equivalent +50 D. sph.	
(10)	+0.50 D. sph.	0 D. cyl.	= equivalent +50 D. sph.	

On trial of these formulas, formulas 3, 4, or 5 are likely to be accepted, with vision comparable to the full correction. One, preferably formula 5, may well be worn with comfort.

I do not advocate routine partial correction for astigmatism. In selected cases, however, it is useful, especially when performed in the manner described.

Astigmatic dials and the ophthalmometer are not used universally by American ophthalmologists. Many excellent refractionists use them as a routine, and others do not use them at all. Both methods have a definite place in the armamentarium of the refractionist. I believe that the ophthalmologist should be perfectly familiar with all methods and tests, so that he can employ each to its best advantage in a given circumstance. I often use a dial for a quick check of uncorrected or improperly corrected astigmatism at the beginning or conclusion of a refraction. When one gazes intently at a dial for a few minutes, the intensity of the lines begins to shift and vary with fatigue, and the dial then becomes an optical illusion. Therefore, I do not like it for prolonged or refined testing. The ophthalmometer has been too inaccurate, in my hands, for routine use. I do employ it, however, rather often in the following exceptional cases: (1) in cases of congenital coloboma of the macula in which there is no retinoscopic reflex and vision is so low that subjective testing is uncertain; (2) for children with a high degree of astigmatism, to help confirm observations on the strength and axis of the cylinder; (3) at original visits, to begin the manifest test so that I may avoid a postcycloplegic test; (4) in troublesome cases in which the condition is obscure or doubtful, in which I wish all the information obtainable; (5) for prepresbyopic and presbyopic patients, after a manifest refraction, to help decide whether to use a cycloplegic (the ophthal-

mometer may indicate that all the astigmatism has not been uncovered); (6) in a routine examination of the eyes, to help decide whether or not a refraction should be done; (7) for the psychologic effect of the instrument on the patient; (8) to differentiate between corneal and lenticular astigmatism, and (9) to determine a doubtful axis in cases in which even with astigmatism of 0.5 D. the axis varies from 90 to 180 degrees in the cycloplegic and postcycloplegic tests.

It seems unnecessary to remark on the indispensability of the cross-cylinder in determining the amount and the axis of astigmatism. Apparently, however, the excellent articles of Jackson [8] and Crisp [9] are not familiar to all. I urge a careful reading of these articles and an earnest trial of this device. Once used, it will never be discarded. Although I accept the method as described, there are several special points which might be emphasized. For determination of the axis in ordinary errors I prefer the weaker cross-cylinders, those of 0.12 and 0.25 D., so that the vision will not be blurred too much. With the retinoscopic findings in place, a preliminary axis is found. Subjectively the spherocylindric relations are adjusted, and the best vision is obtained. As a final procedure the axis is checked again with the cross-cylinder. The axis often will shift 5 degrees at this point, and I believe this final axis taken with the best combination of lenses and keen vision is the correct one, although it may differ from the axis obtained at the preliminary examination. When the spherocylindric relationship has not been worked out accurately, a wrong axis may be found. In using the cross-cylinder for determining the amount of astigmatism, one is likely to obtain an overcorrection or an undercorrection. This appears to be a phenomenon of the interval of Sturm and is attributable to an improper relation between the sphere and the cylinder. If the cross-cylinder appears to lead one too far from the retinoscopic cylinder, the sphere should be rechecked at each change indicated by the cross-cylinder. In other words, the optical effect on both the sphere and the cylinder by the cross-cylinder should be kept in mind when using it for strength. When it is used for strength the cross-cylinder alters both the sphere and the cylinder, and this alteration should be visualized. If the cylindric effect alone is thought of and the cylinder is changed without the sphere being checked, an increasingly distorted image will be produced, and the interval of Sturm will be exaggerated instead of corrected. With these facts in mind, the cross-cylinder can be employed satisfactorily in most cases for finding the amount of astigmatism. There are, however, exceptions

8. Jackson, Edward: Principles Applied in Use of Cross Cylinders, Am. J. Ophth. **12**:897-901 (Nov.) 1929.

9. Crisp, W. H.: The Cross-Cylinder Tests, Especially in Relation to the Astigmatic Axis, Tr. Ophth. Soc. U. Kingdom **51**:495-514, 1931.

to the accuracy of the findings determined with the cross-cylinder for both the axis and the strength, and these findings must be checked by other methods.

In corrections for anisometropia and for antimetropia one should bear in mind the prismatic effect produced as the eyes look from distant to near objects. There occurs a shift of the optical center of the lens upward which produces the effect of a prism base up in hyperopia and that of a prism base down in myopia. This depends on the difference in the power of the two lenses in the vertical meridian. As the eyes are directed downward to read they are looking through the lenses 5 to 8 mm. below the optical centers of the lenses. If the lenses are of equal power in the vertical meridian, the prismatic effect is equal in each and is of no significance. This is not so, however, when the lenses are of unequal power vertically. The prismatic power produced in each lens is figured by the equation $A = P \times D$, in which A is the amount of prism produced, P is the power of the lens in the vertical meridian, and D is the distance in centimeters that the eye looks through the lens below its optical center. The difference in the prismatic power so produced in the two lenses represents the amount of vertical prism which the eye is asked to overcome while reading. To offset this, compensated lenses have to be ordered; for the nonpresbyope these consist of reading glasses with compensating prisms, and for the presbyope, of either reading glasses or special bifocal glasses with the addition of a prism. In spite of the theory, I do not think that compensated lenses are necessary in the majority of such cases, as the patients seem to adjust or tolerate their unequal corrections comfortably. One should bear this problem in mind, however, for the persistently uncomfortable anisometrope and give him the benefit of trial with compensated correction. Incidentally, I see no valid reason why in most cases of anisometropia and antimetropia the error should not be fully corrected eventually. In my experience the patients respond nicely to such correction.

One should consider also the effective power of lenses. The power of strong lenses, such as corrections for aphakia, varies considerably as their distance from the eye is changed. There may be a difference as great as 15 mm. anteroposteriorly in the position of lenses set in the trial frame and their position when they are worn by the patient in his glasses. The equation, $C = KSD^2$, is used to determine the change in power produced by moving a lens forward or backward in front of the eye. C is the amount of change produced in diopters; K is the constant, which is 0.001 when millimeters and diopters are used; S is the distance moved in millimeters, and D, the dioptric power of the lens. Thus the power of a 10 D. lens moved 10 mm. is altered 1 D. This problem, of course, is important only for lenses of higher power.

Ophthalmologists have had a long-standing aversion to the inclusion of prisms in prescriptions for glasses. It has been thought that their use would tend to exaggerate still further the muscular imbalance and cause the eyes to become dependent on them. Further, it has been thought that the amount of prismatic power necessary would increase as time went on. There is, of course, some truth in these ideas, and yet I believe prisms should be prescribed for wear when indicated. All ophthalmologists know that the wearing of correcting lenses for hyperopia and myopia makes the eyes dependent on them and that these corrections have to be increased as time goes on. Still I do not think that for these reasons correcting lenses should not be ordered. Increased visual efficiency and comfort demand these corrections. Likewise, it seems that a prismatic correction should be prescribed for the same reasons, namely, increased visual efficiency and comfort, whenever indicated. I have the feeling also that prisms are not prescribed more generally because of the lack of clear understanding of their action when they are placed in front of the eyes. Optically, one is taught that rays of light are deflected toward the base of a prism as they pass through it. Clinically, it is more useful to remember that the image of an object looked at through a prism is displaced toward the apex of the prism. Prisms are used for two purposes: (1) to displace or measure the displacement of the ocular images and (2) to make the eyes follow as far as possible displaced images. The first use is, of course, for the measurement or correction of deviations, diplopia or phorias. The second is for determining ductions or for exercise. Again one must understand the behavior of false images. These are found in the direction opposite to the deviation or the tendency to deviate of the eye because there has been a shift in the retinal pattern of the deviating eye without cerebral recognition of the fact. The brain continues to project the impulse received as though the eyes were directed normally. Therefore, in using prisms one either has or creates a false image, and prescription becomes simply a matter of knowing in which direction one wishes to displace the image and recognition of the fact that a prism displaces the image looked at toward its apex. When thus visualized, the use of prisms is very simple and also very useful for both diagnosis and prescribing.

In prescribing prisms I prefer to use a prism of the minimal degree with which single vision can be obtained. There is often considerable spread between the minimal and the maximal prism accepted. Other things being equal and the amount of prismatic power small, I prefer to place the prism before the nondominant eye. If the degree of the prism is more than 3, I prefer to divide it between the two eyes. To determine the minimal prismatic requirement, binocular fixation can be

fatigued by measuring the phoria slowly and bringing out the latent tendency to see double. Then before measuring ductions, the diplopia, if apparent, can be corrected by the minimal amount of prismatic power. Repeated tests of this character often help to find the amount of prismatic power necessary for single vision, for distant and for near objects. The amount of any such prism required must be added to or subtracted from the duction powers subsequently found. When vertical deviation differs for distance vision and for near vision (and it often does) one should not correct more than the lesser finding in glasses to be worn both for distance vision and for near vision. Otherwise, the eyes may be corrected for one distance and overcorrected or undercorrected for the other. If one wishes to give a different prismatic correction for distance vision and for near vision, either two pairs of glasses or special bifocal glasses are necessary. In cases of a combined lateral and vertical deviation the vertical deviation should perhaps be corrected first, although often both should be corrected, to give balance. Prisms seem to be most useful in the treatment of the insufficient convergence, divergence and vertical divergence and, conversely, least effective in the treatment of the excesses of these functions. Before prescribing prisms one should be sure that any existing ametropia has been corrected carefully and balanced vision has been obtained for both distant and near objects. Especially is this true in cases of anisometropia and antimetropia. Often when this has been done, apparent imbalances and symptoms disappear. If muscular imbalance and symptoms persist, however, the discriminating use of prisms seems to be next in line for consideration.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

PRESENCE OF GANGLION CELLS IN THE CILIARY NERVES OF THE ORBIT AND THEIR HISTOLOGIC SIGNIFICANCE. J. MAWAS, Bull. Soc. d'opht. de Paris, February 1936, p. 89.

The structure and rôle of the ganglion cells which form the compact mass of the ciliary ganglion have been well described. One may readily understand their function and final distribution. However, the presence of the ganglion cells in the ciliary nerves of the orbit needs histologic and physiologic elucidation. The problem of the accessory ganglions reported by Foesbeck, Svitzer, Antonnelli, Laffay, Fuchs and Axenfeld is not drawn into this question, but the author is concerned with the massed and isolated ganglion cells seen in the interior of the ciliary nerves. The classic textbook of Schieck and Brückner and that of Duke-Elder mention only their existence, but they were reported by Reichart in 1875 and, more recently, by Gallemaerts in 1899, André-Thomas in 1910, d'Erchia in 1895 and Cannieu in 1899. Mawas studied first the distribution of the ganglion cells with the nerve fibers. In the trigeminal and oculomotor nerves ganglion cells are noted in the terminal fillets of the nerves. The presence of the cells in the ciliary ganglion must be the origin of such cells in these nerve fibers. It is felt that such cells take the place of the sympathetic nerves to these parts.

L. L. MAYER.

Conjunctiva

THE HISTOPATHOLOGY OF PARINAUD'S CONJUNCTIVITIS. H. D. LAMB, Am. J. Ophth. **19:** 571 (July) 1936.

Lamb gives a brief review of the literature on Parinaud's conjunctivitis, starting with the original article in 1889 and ending with a description of the pathologic changes noted by Verhoeff, who first discovered Leptothrix in the lesions. Two cases are reported. The following summary is given:

"The clinical reports and histologic findings with the presence of Verhoeff's leptothrix are described in two cases of Parinaud's conjunctivitis. In both cases, an eosinophilia of 4 percent was found."

W. S. REESE.

INTERESTING CASE OF SYPHILIS OF THE PALPEBRAL CONJUNCTIVA. PAULINA SATANOWSKY and P. KURLAT, Arch. de oftal. de Buenos Aires **11:** 533 (Sept.) 1936.

This is the report of a case of an ulcerative disease of the palpebral conjunctiva, accompanied with swelling of the preauricular glands and

followed by secondary lesions of the throat. The patient gave a history of syphilis four years before. The authors, after a full discussion of the different elements in the case, express the opinion that the condition was a primary lesion (due to reinfection after a prior cure).

C. E. FINLAY.

A CASE OF PEMPHIGUS OF THE CONJUNCTIVA. D. M. BAKHIR, Sovet. vestnik oftal. **9:** 920, 1936.

This case is reported because of the rarity of the condition.

A young healthy person had pemphigus of the urethra, with formation of a stricture which took a malignant course, as the artificially made fistula also became stenosed. There was pemphigus of the conjunctiva of the lower fornices of each eye, with scar formation. The mucous membrane of the mouth (the palate) was also affected.

O. SITCHEVSKA.

Congenital Anomalies

OCULAR NEVUS. D. ARGÜELLO and C. VON GROLMANN, Arch. de oftal. de Buenos Aires **11:** 417 (July) 1936.

Argüello and von Grolmann first give the results of their studies of melanosis and the origin of pigments in the body—(1) hematogenous, (2) cellular (lipochromes and melanin), (3) metallic and (4) rhodopsin. Melanin seems to originate in a biologic adaptation to radiant energy and is especially abundant in tropical races. The authors discuss the different racial types and the distribution of melanin in different ocular tissues and report the case of an Argentine with lesions of the skin of the lids and temple, deposits of melanin in the conjunctiva, sclera and iris and a semilunar area of pigmentation of the disk, without any depreciation of vision.

C. E. FINLAY.

MULTIPLE PIGMENTED CONGENITAL CYSTS OF THE IRIS. J. A. SENÁ, Arch. de oftal. de Buenos Aires **11:** 462 (Aug.) 1936.

After giving a detailed classification of cysts of the iris according to their origin, with abundant bibliographic references, Sená stresses the production of spontaneous interepithelial cysts due to nonfusion of the two layers of the epithelium of the iris and reports a case of multiple cysts of this nature near the pupillary margin in each eye. A stereophotograph and a colored drawing illustrating the condition are included.

C. E. FINLAY.

THE OPTIC VESICLE, COLOBOMAS OF THE FUNDUS OCULI AND OTHER MALFORMATIONS. C. S. DAMEL and E. ADROGUÉ, Arch. de oftal. de Buenos Aires **11:** 473 (Aug.) 1936.

This is a lengthy paper, which does not lend itself to abstracting, in which the authors review at length the literature on the subject given in

the title. They discuss different points of view, classifications and the mode of production of the malformations. The article is illustrated with numerous cuts and photomicrographs, many of original preparations.

C. E. FINLAY.

Cornea and Sclera

THE ASSOCIATION OF DENDRITIC ULCER OF THE CORNEA AND OF SUPERFICIAL PUNCTATE KERATITIS WITH HERPES FACIALIS. H. NEAME, Brit. J. Ophth. **21:** 298 (June) 1937.

The first patient, a man aged 49, when first seen had a sharp febrile attack, a marked herpetiform eruption of the face and fairly extensive dendritic ulceration of the cornea in two areas, one small and the other large. The second patient was a man aged 29 with pulmonary tuberculosis, which had been treated by artificial pneumothorax. Associated with the condition of the chest there was herpes facialis. Later there was a dendritic ulcer of the cornea, with two spots that were classed as nummular or macular keratitis. The third patient was a man aged 28 with typical superficial punctate keratitis, with two small dendritic ulcers near the margin of the cornea. The condition was associated with herpes facialis.

Neame discusses the pathogenesis, etiology, symptoms and signs of the superficial type of corneal ulcers.

The article has three illustrations.

W. ZENTMAYER.

SYPHILITIC LESIONS OF THE CORNEA AND THE CONJUNCTIVA IN A NEW-BORN INFANT. NICOLAS, P. BONNET, GONNET, BANSILLON and COLRAT, Bull. Soc. d'opht. de Paris, February 1936, p. 97.

In the corneal ulcers of a premature infant the spirochete was observed. The Wassermann reaction of the blood had been negative. In the first few days the infant showed conjunctivitis of each eye, which was more marked in the left eye, with edema of the lids, chemosis and staphylococcic suppuration. Ulcers of the corneas were present, and, in addition, interstitial infiltration with vascularization. Consultation with Professor Nicolas, of the department of diseases of the skin, resulted in a diagnosis of syphilis, and the treponema was observed in corneal scrapings. The question of hereditary syphilis is discussed. The liver and the spleen were not enlarged, and there were no buccal lesions. No chancre was found in the examination of the passages. An isolated enlarged gland was found in the maxillary angle. This was very hard. General treatment, consisting of mercury rubs, was given. Recovery was immediate. The mode of contamination remains obscure. The Wassermann reaction of both parents was negative.

L. L. MAYER.

CORNEAL ULCERS OBSERVED AFTER CERTAIN INJURIES IN PREGLAUCOMATOUS EYES. A. VON NAGY, Klin. Monatsbl. f. Augenh. **97:** 523 (Oct.) 1936.

Nagy reports the case histories of three patients with apparently irrelevant corneal injuries followed by obstinate corneal ulcers and

glaucoma later on. These patients and others with a similar course of their injuries were of advanced age, and, considering their injuries slight, they neglected them for a week or longer. The injuries of the three patients in question were caused by a splinter of rock, a chip of wood and a darning needle, respectively. The intra-ocular tension was raised on admission to the polyclinic. Nagy thinks that these eyes were predisposed to glaucoma, which became manifest probably as a result of dilatation of the ciliary vessels by means of irritation of the sympathetic nerves. A number of publications in point are quoted, and the result of Ehrlich's experiment with fluorescein, applied in these three cases, is described. The author arrives at the following conclusions: 1. Mydriatics must be used with caution in cases of traumatic corneal ulcers in patients of advanced age. 2. Posterior sclerotomy should be done at an early date. 3. An attack of glaucoma occurring in one of these patients later on is clinical proof for the correctness of the view arrived at regarding the glaucomatous diathesis. 4. Correct evaluation of the complaints may be of preventive importance for both eyes.

K. L. STOLL.

CALCEMIA AND CALCIUM THERAPY IN SO-CALLED ECZEMATOUS BLEPHAROKERATOCONJUNCTIVITIS. D. LĂZĂRESCU and E. DAMIAN, Klin. Monatsbl. f. Augenh. **97**: 626 (Nov.) 1936.

Lăzărescu and Damian discuss the relation of tuberculosis to the type of blepharokeratoconjunctivitis which is inadequately called "eczematous." This disease occurs frequently in persons with latent tuberculosis, although the bacillus of Koch could never be found in phlyctenules. The research on calcium deficiency as a cause of edema and other disturbances is evaluated, and the bibliography in point is cited. Data are given relative to the calcium content in the protoplasm of the cell, the vitreous, the blood serum and elsewhere in normal conditions and pathologic conditions and in pregnancy. Among the divers actions of calcium given therapeutically, its sedative action in some ocular diseases is referred to as described by Georgiana Theobald.

The authors examined the calcium content of seventy persons, using the method of Kramer and Tisdall. These patients suffered from divers ocular diseases, forty from fulminant eczematous keratoconjunctivitis or blepharokeratoconjunctivitis, which was combined with phlyctenules in most of them. Tracheobronchial adenopathy was roentgenologically found in nine children who, however, had no pulmonary lesions. The blood for the tests was taken after the first breakfast. The calcium content of the normal persons varied between 92.21 and 112 mg. per thousand cubic centimeters of serum, with an average of 100 mg., except for pregnant women, for whom the average was 88.8 mg. In seventeen patients with eczematous conjunctivitis the values varied between 83 and 89 mg. These patients received local treatment and 10 cc. of calcium intravenously every forty-eight hours. The calcium content increased with the treatment. The duration of the treatment was seventeen days, and no untoward effects of the injections were noted. Calcium gluconate and calcium thiosulfate administered intramuscularly or intravenously not only promptly reduce photophobia,

spasms and edema but also accelerate recovery, with reduction of hospitalization to one half, and incidentally improve the general physical condition of the patient. Intravenous injections act more promptly, intramuscular injections more protractedly. The settling time of the erythrocytes, which is accelerated in eczematous keratoconjunctivitis, is retarded by this treatment, as a result of general physical improvement.

K. L. STOLL.

Experimental Pathology

THE EFFECT OF GAMMA RAYS ON CELL DIVISION IN THE DEVELOPING RAT RETINA. K. TANSLEY, F. G. SPEAR and A. GLÜCKSMAN, Brit. J. Ophth. **21:** 273 (June) 1937.

The object of the experiments undertaken by the authors was:

1. To determine the precise effect of gamma irradiation on the division of cells in mammalian tissue.

2. To compare the results with those previously obtained with chick tissue.

3. To determine quantitatively the alteration in division of cells and the production of cellular degeneration after different doses of radiation.

4. To investigate the relationship, if any exists, between the mitotic changes and the degeneration which follows irradiation.

The following summary is given:

"1. A method of exposing the developing rat eye to fairly accurate doses of gamma radiation is described.

"2. It has been found that the normal 50:100:50 value for the prophase: metaphase: telephase ratio is altered as a result of irradiation. The distortion of the ratio varies with the size of the dose and the time-interval between irradiation and examination of material.

"3. A relationship has been established between alteration in cell division and the occurrence of degeneration among undifferentiated cells.

"4. The results show that cellular degeneration can be produced by very small doses of gamma rays and that this degeneration is accounted for by an effect upon cells which are about to divide which unfits them for the ordeal of mitosis.

"5. The quantitative results obtained from this mammalian tissue closely resemble those already provided by experiments on avian fibroblasts cultivated *in vitro*.

"6. A statistical analysis has been made of the individual variation of mitotic counts in different rats, both irradiated and unirradiated."

W. ZENTMAYER.

General

THE INTRA-OCULAR TENSION AND PREGNANCY. E. DE GRÓSZ, Ann. d'ocul. **174:** 167 (March) 1937.

The intra-ocular tension depends on a number of factors: the pressure of the intra-ocular vessels, the permeability of these vessels, swell-

ing of the vitreous and filtration of the aqueous. The regulation of these functions is governed by the vegetative centers, which are under hormonal influence.

Among 50 pregnant women examined, [illegible]l and Imre found the following results: In 42 the tension was diminished; this condition existed particularly in those who had acromegaly. In 2 the tension was normal, and in 6 it was increased.

De Grósz himself observed 105 patients, and in this group he made 151 examinations. The results are set forth in a number of tables and charts. A bibliography is given.

S. H. McKee.

New Method of Anesthesia and of Elective Paralysis of Various Nerves and Muscles of the Orbit. C. Dejean, Arch. d'opht. **53:** 32 (Jan.) 1936.

Dejean points out as an objection to massive orbital injection the fact that the eye is immobilized and turned up, which is a poor position for most operations. The purpose of his study has been to devise a means of paralyzing one muscle or one group of muscles so that the eye will be turned in the direction desired. He first describes in detail a method of injecting the infra-orbital nerve in its canal. For obtaining paralysis of the superior rectus muscle a 5 to 6 cm. needle is used to puncture the skin from 3 to 4 mm. below the external angle of the lids; this passes round the globe and is carried backward and upward in the direction of the parietal boss of the opposite side. The injection is made when the needle has entered a depth of from 3 to 5 cm. The relations of the other rectus muscles and the nerves are described, and the procedures necessary to affect them are given. The conditions necessary for their elective paralysis are (1) a small amount of the fluid for injection, (2) injection back of the fibrous sheath of the muscle and (3) injection as nearly as possible where the nerve enters the muscle. These relationships are all given in detail.

S. B. Marlow.

General Diseases

Dental Focal Infection in Ophthalmology. C. Charlin and L. Gomez V., Arch. de oftal. de Buenos Aires **11:** 251 (May) 1936.

This paper is based on the findings in fifty cases taken from Charlin's clinical records and from the literature, in all of which the ocular lesion was resistant to the usual therapeutic measures and in which spectacular improvement followed destruction of the dental focus.

The ocular lesions fell under the following headings: conjunctivitis, three cases; keratitis (vesicular, three cases; ulcerative, three cases; parenchymatous, two cases) eight cases; karato-iritis, one case; episcleritis, one case; iritis, ten cases; iridocyclitis, four cases; cyclitis, two cases; solitary retinal hypertension, one case; retinal angiospasm, one case; neuroretinitis, four cases; retrobulbar neuritis, three cases; oculomotor paralysis, five cases; orbital cellulitis, one case; nasal syndrome, two cases, and glaucoma, one case.

As a rule, the ocular disease is irregular in its course, with abrupt exacerbations and remissions, and the clinical and laboratory findings are negative.

The lesion was unilateral in 94 per cent of the cases.

Details of the ocular lesions are noted.

As regards the provocative dental lesion, in thirty-four of the fifty cases it affected a single tooth; in forty-seven cases it was painless, and almost invariably the affected tooth was an obturated tooth with 4 degree caries; in most cases the affected tooth had multiple roots. In most cases in which a single root was affected the tooth was in the upper maxilla of the same side as the ocular lesion.

The dental focus is produced by an anaerobic micro-organism; the ocular lesion contains no germs.

A detailed abstract of the fifty cases closes the paper.

C. E. FINLAY.

General Pathology

THE CAPILLARIES IN THE NAIL BED IN DISEASES OF THE EYE. S. MIELKE, Arch. f. Augenh. **110:** 236, 1936.

In a large number of cases of diseases of the eye the capillaries of the nail bed were examined with the microscope. Mielke came to the following conclusions:

1. Capillary hemorrhages in the nail bed, as distinguished by Marchesani as an important sign of vascular disease, are frequent.

2. The frequency of these hemorrhages does not run parallel with age, even though arteriosclerosis plays a rôle.

3. Such hemorrhages were found more frequently in persons with myopia, especially myopia with degenerative changes, and in young patients with paralysis of ocular muscles in whom neurologic examination showed no signs of tabes or multiple sclerosis.

4. In cases of inflammation of the uvea there was no special frequency of hemorrhages in the nail bed, and the same was true in cases of glaucoma. The investigations in the patients with glaucoma were not sufficiently extensive to enable one to form any definite conclusions.

F. H. ADLER.

Glaucoma

VARIATIONS IN A FILTRATING CICATRIX IN ONE EYE IN THE COURSE OF GLAUCOMA OF THE UNOPERATED OR FELLOW EYE. J. SÉDAN, Bull. Soc. d'opht. de Paris, February 1936, p. 91.

A curious variation was seen in an eye which had been successfully operated on for glaucoma by the Lagrange technic in 1931 when chronic simple glaucoma developed in the fellow eye in 1935. The right eye, which had been operated on in 1931, had manifested glaucoma after hypertension and uremia. The operative scar, 2 mm. in diameter, showed no change, and the tension of the eye remained 20 mm. of mercury. In May 1935 the patient had profound anemia, the onset of

which was followed by violent pain in the left eye. Acute glaucoma supervened, which was relieved by miotics, with recovery of the normal tension and normal visual acuity. Sédan noted that the filtrating scar of the right eye increased in size fourfold and presented edema of the upper bulbar conjunctiva. He was not interested in the tension, fields or vision of this eye. On relief of the acute condition of the left eye the signs in the right eye subsided. The hypothesis of a general reaction with specific toxic effects of a sympathetic nature is strengthened by this phenomenon.

L. L. MAYER.

Lens

INTRACAPSULAR EXTRACTION OF CATARACT BY THE HUNGARIAN METHOD. G. PARTE, Ann. d'ocul. **174:** 248 (April) 1937.

Parte's intention is not to show the advantages of extraction of cataract in its capsule over the classic method of extracapsular extraction but to draw the attention of ophthalmologists to the operative procedure of intracapsular extraction by a forceps, which is simpler than the procedure generally adopted and which, referring to Emil de Grósz, of Budapest, he calls the Hungarian method.

After a description of the preparation, the retrobulbar injection and a stitch for security, the incision and the iridectomy (the pupil having been previously dilated) are described. The capsule of the lens is grasped near the inferior extremity of the vertical meridian. The zonule of Zinn is then progressively broken, and the lens is extracted by executing an interior version in such a fashion that the inferior part of the equator is brought to the corneal surface. Multiple sutures are used.

Parte has reported this method in the hope that ophthalmologists who have not already done extraction of the lens in the capsule will try it.

S. H. MCKEE.

LENTICULAR ANTIGEN THERAPY. I. BORSOTTI, Ann. di ottal. e clin. ocul. **64:** 744 (Nov.) 1936.

The literature on lens protein, including the clinical reports of Davis, Marquez and others, is reviewed. Borsotti believes that the reports of Davis on the use of lens antigen in incipient cataract are without any sound basis, but that the work on the absorption of cortex after discission in patients given lens antigen must be considered more seriously. In attempting to repeat the work of Biffis and Quaglio he found that after discission in guinea-pigs the results did not permit exact observation, since the amount of cortex in the anterior chamber varied. Instead, he injected an emulsion of beef and rabbit lens so as to fill the anterior chamber after removal of the aqueous. Each of fifty rabbits was given such an injection, and half of them were also each given a subcutaneous injection of beef lens either previous to or after the injection into the anterior chamber. The animals were divided into groups according to various conditions of immunization. Those animals which received subcutaneous injections after the injections into the anterior chamber showed no difference in speed of absorption from the controls. Some of

those which had received the preliminary course of injections showed more rapid absorption (eleven animals). Phenomena of sensitization were seen in this group, however, as shown by a marked inflammatory reaction when injection was made into the anterior chamber. While the subsequent injection did not hasten absorption, it apparently lessened the inflammatory reaction due to the presence of lens material in the anterior chamber. Borsotti concludes that his work does not encourage the use of lens antigen to hasten absorption of cortex but indicates that this may involve risk of provoking an anaphylactic reaction at the time of operation. He believes passive immunization with an immune serum would be free from this danger and should be investigated.

A bibliography is included.

S. R. GIFFORD.

PROLAPSE OF VITREOUS AND GLAUCOMA AFTER INTRACAPSULAR EXTRACTION OF CATARACT. R. F. PEREIRA, Arch. de oftal. de Buenos Aires **11:** 409 (July) 1936.

After reference to a former paper on prolapse of vitreous followed by glaucoma after discission of a secondary cataract, Pereira reports two cases in which there was a similar complication after intracapsular extraction of cataract. In one case the condition followed a violent physical effort, and in the other it occurred spontaneously after pupillary dilatation.

C. E. FINLAY.

Lids

AMYLOIDOSIS OF THE LID. O. I. RYBNIKOVA, Sovet. vestnik oftal. **9:** 911, 1936.

Up to 1928 there were recorded in European and American literature only one hundred and thirty-three cases of amyloidosis of the lid.

A man aged 63 complained of gradually increasing swelling of the right upper lid. Examination revealed marked ptosis of the right eye; the lid was thickened and had a normal skin with smooth folds, and the margin of the lid was S shaped. A tumor about 1 cm. wide could be palpated in the lid; it was elastic, painless and movable and had a rough surface. It was impossible to evert the lid. There was a small round tumor of the scleral conjunctiva at the inner angle. There was present trachoma of stage 3 with pannus of the cornea. A biopsy confirmed the clinical diagnosis of amyloidosis. Extirpation of the upper tarsus with consequent transplantation of the mucous membrane of the lid was done. The ptosis disappeared shortly after the operation, and the lid became normal.

A detailed description of the pathologic process and of the various staining methods used is given. The outstanding feature was the abundance of homogeneous large and small masses with homogeneous fibers between them.

The clinical, pathologic and physiochemical properties of amyloid are described. Various theories of the etiology of amyloidosis are discussed. The association of deposits of amyloid with trachoma and the predisposition of chronically irritated tissues to amyloidosis is noted.

O. SITCHEVSKA.

Neurology

GENICULATE NEURALGIA (NEURALGIA OF THE NERVUS FACIALIS): A FURTHER CONTRIBUTION TO THE SENSORY SYSTEM OF THE FACIAL NERVE AND ITS NEURALGIC CONDITIONS. J. RAMSAY HUNT, Arch. Neurol. & Psychiat. **37:** 253 (Feb.) 1937.

Although this article is primarily concerned with neuralgia arising from the geniculate ganglion, nevertheless the discussion of the anatomic features of the sensory and sympathetic nerves of the face and their relation to deep orbital pain should be of interest to all ophthalmologists. There is a discussion of sphenopalatine ganglion neurosis (Sluder's syndrome), the pain of which is localized in the root of the nose, in and about the eye and in the upper part of the face and the upper teeth and sometimes also in the lower jaw and the lower teeth, in the temple and about the zygoma, the ear, the mastoid, the occiput and the neck, the hand and even the finger tips.

Hunt summarizes the neuralgic disorders of the various cranial nerves as follows:

1. True trigeminal neuralgia, which is distributed in one or more branches of the trifacial nerve and in which the pain is localized in the more superficial structures of the face and intra-oral region. This is classic prosopalgia or trifacial neuralgia. In cases of neuralgia of the third division of the fifth nerve there is often associated otalgia.

2. Geniculate neuralgia, which involves the deeper structures of the face. This is characterized by pain in the deep posterior orbital, palatal and nasal regions, with painful pressure sensation in the face. This is geniculate deep prosopalgia, and with it there is associated geniculate otalgia.

3. Glossopharyngeal neuralgia, which is characterized by neuralgic pains in the distribution of the glossopharyngeal nerve at the base of the tongue and the adjacent regions of the throat and by associated otalgia.

4. Superior laryngeal neuralgia, of vagal origin, in which the pains are localized in the region of the larynx, with associated otalgia.

All these various forms are accessible to surgical intervention by the cranial method of approach through the posterior fossa, which exposes the fifth, seventh, ninth and tenth cranial nerves. If this procedure is carried out with the use of local anesthesia, it is possible by touching any one of the nerves to reproduce the neuralgic pain, thus confirming the clinical diagnosis, which is often involved and difficult.

An excellent series of references is included.

R. IRVINE.

CERTAIN REACTIONS OF THE OCULONASAL NERVES. L. HALBRON, Ann. d'ocul. **174:** 145 (March) 1937.

The related reactions of the eye and the nose, both physiologic and pathologic, have long been known. Simultaneous stimulation of the organovegetative system of the orbit and that of the nasal fossae causes sensory and vasomotor syndromes which are no more pituitary of ocular origin than orbital of nasal origin. Halbron purposely does not discuss suppurative sinusitis and the orbital complications.

In the section on the clinical study of oculonasal nervous reactions the author discusses (*a*) the syndrome of the nasal nerve, reporting two cases observed by him in which the symptoms of pain, ocular disturbances and acute rhinitis occurred, and (*b*) the syndrome of the sphenopalatine ganglion, the symptoms of which are pain and involvement of sympathetic nerves.

In the section on diagnosis of the syndrome of the nasal nerve and that of the sphenopalatine ganglion he discusses these syndromes and then the clinical diagnosis of the two syndromes in relation to various types of facial neuralgia. The last topic is subdivided into neuralgia of facial nerves, pain in the ophthalmic zone and neuralgia of facial sympathetic nerves.

Treatment of these syndromes often proves their spasmodicity and is therefore of diagnostic value. Local treatment recommended is the introduction of cocaine into the nostril; general treatment consists in soothing the nervous system and attempting to eliminate intoxications or endocrine disturbances.

S. H. McKee

Operations

The Fluorescent Lamp for Cataract Surgery. H. R. Hildreth, Am. J. Ophth. **19:** 770 (Sept.) 1936.

Hildreth describes the fluorescent lamp and enumerates its uses as a therapeutic and diagnostic agent and for photography of the eye and surgery of the lens. He emphasizes the latter.

W. S. Reese.

Marginal Cutaneous Blepharoplasty in Palpebral Repairs. L. Dupuy-Dutemps, Ann. d'ocul. **174:** 312 (May) 1937.

In cutaneous blepharoplasty, when the loss of substance is not extensive, the edge-to-edge tarsorraphy usually employed gives sufficient exposure on the surface to allow a good result. However, in cases in which there is strong fibrous retraction and the lid has to be reconstructed in the greater part of its width, this method is insufficient. The opposite palpebral border in these circumstances has been a good deal lowered and usually has an inflamed surface. The result of an attempted blepharoplasty under these circumstances remains imperfect, and although the lagophthalmus and ectropion may be lessened, they still persist. This insufficiency is met with especially in cases in which extensive repair of the lower lid is required and particularly if the eye is missing or has become atrophic.

Dupuy-Dutemps describes in detail, supplying four illustrations, an operation which he considers satisfactory, in the conditions already described.

S. H. McKee.

First Series of Partial Penetrating Corneal Transplantation. I. F. Kopp, Sovet. vestnik oftal. **10:** 347, 1937.

Kopp performed thirty corneal transplantations from March 1934 to March 1937 after von Hippel's, Elschnig's and Filatov's method. The first eleven patients whose cases are reported here were followed

from twelve to eighteen months. In five of these the transplant took and was transparent, and vision was improved from counting of fingers to 0.1 (three cases), 0.2 and 0.4, respectively.

A detailed analysis of the cases, a description of the postoperative course and the results of histologic examination are given. Successful transplantation was observed, first, in those cases of leukoma in which there was a sufficient amount of sound corneal elements spread over a wide operative field, second, in those cases in which the anterior chamber was present and, third, in those cases in which the process which caused the corneal opacity was completed, particularly in cases of trachomatous pannus. The injury of the endothelium and the formation of folds in Descemet's membrane of the transplant favor the production of fibrinous exudate, with resultant vascularization and connective tissue scarring. Kopp injects epinephrine hydrochloride subconjunctivally forty minutes before the operation in order to keep the pupil dilated. Traumatization of the lens occurred only once. Kopp considers Filatov's hand trephine sufficient and effective for the operation. He recommends the wide use of the cadaver's cornea because the transplant is taken from a healthy eye and because the material can be obtained whenever it is needed.

Corneal transplantation was performed on forty-five rabbits previous to the keratoplasty performed on human beings.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

UNUSUALLY LARGE MUCOCELE OF THE FRONTAL SINUS, WITH SEVERE VISUAL DISTURBANCES AND RECOVERY. F. CSILLAG, Klin. Monatsbl. f. Augenh. **97:** 663 (Nov.) 1936.

The right eye of a woman aged 52 was swollen for about five months prior to observation by the author. The attending general practitioner had tried to empty the tumor by insertion of a cannula into the upper lid. Preceding grip, syphilis and tuberculosis were excluded. The right palpebral fissure was narrowed and displaced downward and temporally; the upper lid showed ptosis and was immovable, cyanotic and crossed by distended blood vessels. No tenderness on touch existed. A massive, tight but barely fluctuating tumor the size of a walnut was seen below the upper lid. It arose from the frontal bone, causing exophthalmos. The upper orbital margin was not palpable. The eyeball was flattened from above. The disk was edematous and prominent; the retinal veins were slightly distended and tortuous, and retinal hemorrhages were absent.

A roentgenogram revealed the upper margin of the orbit greatly deviated upward and the entrance of the orbit and the frontal sinus generally widened, while the periosteum of the temporal upper margin was thickened. An incision was made along the eyebrow; the lid was pulled downward, and the tumor was removed; it burst during extirpation. The mucocele extended backward toward the optic foramen, diverting the optic nerve; the osseous wall of the orbit and an area of the dura mater were eroded. Nevertheless, prompt recovery followed, and vision of 5/10 of normal was regained, with a prospect of further improvement.

K. L. STOLL.

Pharmacology

DOES ERGOTAMINE TARTRATE CAUSE PARALYSIS OF THE SYMPATHETIC NERVE OF THE IRIS? E. FROMMEL and D. ZIMMET, Ann. d'ocul. **174:** 178 (March) 1937.

It is known that ergotamine tartrate acts clinically as if it paralyzed the sympathetic nerves; there is not a neurologist or a cardiologist, who, experimenting with the action of this drug, is not surprised by the quieting qualities of this medicine on the sympathetic nervous system. Frommel and Zimmet have shown that the tachycardia of rabbits which have for many days been given injections of thyroxin or of an estrogenic substance was influenced immediately by ergotamine injected intravenously.

The details of the experiments of the authors are described. They conclude from their studies that the miosis provoked by ergotamine tartrate is not only of a paralytic order, because excitation of the sympathetic nerves produces also mydriasis. The drug does not totally paralyze the ocular sympathetic nerves. Ergotamine tartrate, in other words, does not inhibit the action of epinephrine on the sphincter of the iris, as is the case with the arterial pressure.

S. H. MCKEE.

Physiology

THE SPHENOPALATINE GANGLION AND THE EYE. DUBOIS-POULSEN, Ann. d'ocul. **174:** 217 (April) 1937.

Certain interested advertisers have brought into style reflex therapy, especially that related to the nasal mucosa. It is presented as a miraculous method and a cure-all. In 1925 Halphen showed that these treatments were directed to the sphenopalatine ganglion.

This ganglion possesses numerous connections with the eye, and it is natural to think that the therapeutic successes obtained in the field of general medicine might be repeated in that of ophthalmology.

Dubois-Poulsen reports on the anatomic, physiologic and pathologic aspects of his studies. Under the heading Physiologic Aspects come the following: I. Secretory Activity: Tearing with Stenosis of the Lacrimal Tract. II. Trophic and Vasomotor Disturbance: Lesions of the Anterior Segment of the Eye. III. The Rôle of Sensitivity: Facial Allergies, Glaucoma, Atrophy of the Optic Nerve and Diverse Lesions.

Much pain is referred to the region of the sphenopalatine ganglion, and for this reason study of the ganglion and the relief of pain by anesthesia merits attention. A bibliography is appended.

S. H. MCKEE.

Retina and Optic Nerve

INFANTILE AND CONGENITAL RETINAL FOLD. A. TILLEMA, Brit. J. Ophth. **21:** 94 (Feb.) 1937.

A slight divergent squint had developed in a child 3½ years old, and the eye looked strange. The lens was turbid in its deeper layers, especially at the center. Behind the lens, temporally and below, a fairly well defined mass was seen against the retina. The eye was

removed. Microscopic examination showed a few posterior synechiae. The lens was liquefied and vacuolated in the anterior cortex. Delicate fibers of connective tissue were attached to the posterior surface and ran toward the ora serrata. The retina was completely detached. It showed two folds, one extending from the optic papilla toward the pars plana of the ciliary body, downward and slightly outward, and a second just behind the ora serrata, extending in a circular direction. The crests of both folds were attached to strands of connective tissue. All these fibers joined behind the lens in a fairly vascular mass of connective tissue. The optic papilla was 2 mm. in front of the retina. Many of the retinal rods and cones were missing. Many small spaces were filled with a homogeneous coagulation. Scattered leukocytes were present. The retinal veins were surrounded by closely packed lymphocytes. Close to the large fold the retina exhibited disorganization and a hole. In this region the inflammatory changes were most marked. The optic nerve traversed the subretinal space for a distance of 2 mm. From the optic nerve a strong fibrous band continued forward. It contained many fibroblasts. The retina and connective tissue membrane contained scattered polymorphonuclear leukocytes. The central artery of the retina divided into several branches close to the optic papilla. No remnants of a hyaloid vessel were observed.

The inflammatory changes were confined almost entirely to the retina and vitreous, and for this reason it is not difficult to reconstruct the pathologic process. Retinitis, probably metastatic, was followed by abscess formation in the vitreous. This abscess subsided and was organized. Fibrous tissue then contracted and pulled every structure to which it was attached toward the center of the eyeball. Thus the retina, optic nerve and choroid were displaced.

Tillema concludes that in his case the anomaly was caused by inflammation, probably retinitis; in Weve's eighth case it was probably caused by cyclitis of the pars plana of the ciliary body. It is suggested that in Weve's first, fourth, sixth and seventh cases the condition was related to high myopia. For all other cases an inhibition of normal development of unknown origin must be accepted; for the group a familial occurrence has not been accepted.

The article is illustrated.

W. ZENTMAYER.

DIFFUSE DETACHMENT OF THE RETINA IN A SCROFULOUS MYOPE. BUJADOUX, Bull. Soc. d'opht. de Paris, February 1936, p. 100.

A woman of 38 years consulted Bujadoux because of high myopia. Visual acuity of the left eye was brought to 1.0 with a —7 D. sphere Vision of the right eye was barely 1/2 with the same correction. Examination of the fundus revealed a large white lesion behind the retina in the vicinity of the right macular area, surrounded with a transparent zone. The patient was being treated for Pott's disease by the orthopedic department. Subconjunctival injections of mercury were of no avail. During this treatment new lesions of similar nature appeared in the temporal portion of the retina. Gradually the entire temporal portion of the retina, including the macular area, became detached. Treatment with the methylic antigen resulted in maintenance of vision of 1/2. The detachment persisted but remained the same. The patient was

presented to obtain advice as to the propriety of surgical intervention for the detachment. In view of the probably tuberculous nature of the disease underlying the detachment, Bujadoux feels that a happy termination has been achieved.

L. L. MAYER.

FAMILIAL MACULAR DEGENERATION OF UNKNOWN CAUSE. H. TILLÉ, Bull. Soc. d'opht. de Paris, July 1936, p. 548.

In two young girls, sisters, of a family of eight children, bilateral macular degeneration which had begun apparently at birth had resulted in loss of central vision. Tillé feels that this condition does not fit in with that described as due to syphilis by Morax in France, with that due to tuberculosis as shown by authorities of central Europe or with that caused by abiotrophy or focal infection as described by North American writers. Photographs of the macular regions of both patients are shown. All physical examinations showed no evidence of disease, and the antecedents in two previous generations had normal eyes. Treatment was of no avail. Tillé is unable to classify the lesion in these patients with any reported in the previous literature, which he reviews.

L. L. MAYER.

TRANSITORY BLINDNESS FROM RETINAL ARTERIAL HYPERTENSION. D. CATTANEO, Ann. di ottal. e clin. ocul. **65:** 81 (Feb.) 1937.

During an attack of mushroom poisoning a man of 36 noted sudden loss of vision. Vision was reduced to perception of light in each eye. The retinal vessels were somewhat small, and the disks were slightly pale. A few small hemorrhages were noted. The general blood pressure was 205 systolic and 115 diastolic, and the urine showed albumin and casts. The retinal blood pressure was estimated with Bailliart's dynamometer. In each eye the arterial pressure was 150 systolic and 105 diastolic. The venous pressure in the right eye was 15 mm. and in the left eye 20 mm. Intramuscular injections of acetylcholine had no effect on vision or on the retinal blood pressure. Three days after the onset of the visual symptoms 0.1 Gm. of acetycholine was given by retrobulbar injection. The next day vision had improved to 4/10 in the right eye and 9/10 in the left, and both the general and the retinal blood pressure had begun to diminish. A second retrobulbar injection was given. Five days after the onset of the symptoms vision was normal in each eye. The general blood pressure was 125 systolic and 80 diastolic. The retinal arterial pressure was 100 diastolic and 45 systolic for the right eye and 95 systolic and 40 diastolic for the left. The retinal venous pressure was 5 mm. in each eye. The renal condition cleared up more slowly but completely, while no further loss of vision was noted during the period of observation. Sudden hypertension with associated visual symptoms has not previously been reported as a result of mushroom poisoning but was evidently the condition in this case, since no signs of previous vascular disease were present. Cattaneo believes that a sudden vascular spasm as a result of the poisoning was responsible for the renal and vascular changes. A bibliography is included.

S. R. GIFFORD.

RETINAL ANGIOSCOPY IN ARTERIAL HYPERTENSION AND RETINITIS OF PREGNANCY. C. E. LUQUE, Arch. de oftal. de Buenos Aires **11:** 272 (May) 1936.

Luque stresses the importance of angioscopy in determining whether arterial hypertension appearing in pregnancy is of toxic (eclamptic) origin or is pseudo-eclamptic and the consequence of an aggravation of latent preexisting nephritis and, likewise, whether retinitis coming on in pregnancy is toxic or due to an exacerbation of latent nephritis. These points are easily determined by angioscopy, there being no lesion of the walls of the retinal vessels (best observed at the arteriovenous crossings) in the toxic varieties and more or less pronounced lesions in those varieties complicating chronic nephritis.

Six cases are reported: two of arterial hypertension in pregnancy in which the ophthalmoscopic findings confirmed the toxic nature of the condition as suspected by the obstetrician, two in which a suspected nephritic origin was confirmed and two in which the lesions of the fundus in the one patient and their absence in the other patient furnished the necessary data for a correct diagnosis, the other clinical symptoms pointing in another direction.

C. E. FINLAY.

CYSTIC DEGENERATION OF THE ORA SERRATA. GIOVANNI BRUNO, Arch. f. Augenh. **110:** 183, 1936.

Bruno concludes that cystic degeneration of the retina is not a normal change due to senility, as a number of authors have asserted, but that it is a chronic edema which destroys the layers of the retina. This edema is apparently caused by a change in the permeability of the capillaries at the anterior edge of the retina.

F. H. ADLER.

FERMENTS OF THE RETRORETINAL FLUID IN RETINAL DETACHMENT. H. WEVE and F. P. FISCHER, Arch. f. Augenh. **110:** 198, 1936.

Weve and Fischer collected the retroretinal fluid from a series of patients with retinal detachment at the time of operation. Diathermy needles were used for making the punctures. All the fluid was examined for red blood cells and discarded if any were found. Although there was a possibility of contamination with lacrimal fluid, tests showed that lacrimal fluid itself never contained starch-splitting ferments. In a series of cases it was found that in a number amylase was present, which came from the retina. In another group of cases of detachment of the retina the retroretinal fluid contained no amylase. It was not possible to distinguish clinically between the two groups. Amylase was found in eight of twenty cases.

F. H. ADLER.

BLOOD PRESSURE IN THE CENTRAL RETINAL ARTERY. S. SUGANUMA, Klin. Monatsbl. f. Augenh. **97:** 498 (Oct.) 1936.

This continuation of the first report, which appeared in a previous issue of the *Klinische Monatsblätter für Augenheilkunde* (**96:** 74 [Jan.] 1936; abstr., ARCH. OPHTH. **16:** 880 [Oct.] 1936). In the present

article Suganuma reports on the blood pressure in the central retinal artery in divers forms of general hypertension and in so-called isolated cephalic hypertension. Tables with data on the results of urinalysis, the blood pressure and the appearance of the fundus are included, and an extensive bibliography is appended. The relation of the systolic pressure in the central retinal artery to the general blood pressure was 56: 100 in essential hypertension and 51 : 100 in chronic nephritis and in secondary atrophy of the kidney.

Considerable fluctuations have been observed in essential hypertension. The relation of the pressure of the pulse to the maximal pressure, i. e., the coefficient of the blood pressure, in the retina averages 0.28 in essential hypertension and 0.24 in nephritides. The capillary blood pressure in the macular region of the retina shows an irregular increase in general hypertension, normal values occurring occasionally. The latter result is contradictory to Kylins' readings. Suganuma places under the heading "isolated cephalic hypertension" all the cases in which the general hypertension was normal while the pressure in the retinal arteries was raised. This hypertension was found in divers specific diseases, such as increase of the cerebral pressure, nephritis in pregnancy, epilepsy and retrobulbar neuritis. It occurred, furthermore, in certain stages of essential hypertension and of chronic nephritis. Therefore this hypertension is not rare, but it is readily overlooked, in the author's opinion. Variations of the caliber, reflexes and course of the retinal vessels are not dependent on the height of the blood pressure in the retinal artery. Arteriovenous compression (Gunn's or Salus' phenomenon) is generally due to an increase of the tonus in the wall of the artery, i. e., an increase of the retinal blood pressure. It is especially high in the presence of organic changes in the walls of the vessels. Its degree, however, is not dependent on the height of the local retinal blood pressure. A reliable diagnosis regarding the intra-ocular and intracranial circulation is possible, in the author's opinion, only on measurement of the blood pressure in the retinal vessels, aside from the customary general ophthalmoscopic examinations.

K. L. STOLL.

Trachoma

BACTERIOLOGICAL AND EXPERIMENTAL RESEARCHES ON THE AETIOLOGY OF TRACHOMA. A. CUÉNOD and R. NATAF. Brit. J. Ophth. **21:** 309 (June) 1937.

The authors give the following summary:

"My first researches were made in 1907 in Tunis. They have established that trachoma is a contagious disease, caused by a virus the principal characters of which have been described above.

"Under the influence of researches on typhus and on the Rickettsias in various bacteriological centers, and especially at the Institut Pasteur de Tunis, bacteriological and experimental researches have been made showing that the trachomatous virus has a close relationship with an infra-organism of the Rickettsia family, the presence of which is morphologically demonstrable, as Busacca as well as ourselves has pointed out.

"The anal inoculation of the louse with trachomatous material causes the rapid increase of the organism. After many passages it is still able to reproduce lesions characteristic of trachoma on the conjunctiva of the Barbary ape (singe) and of man.

"Therefore it seems to be proved that trachoma is due to an infection of the conjunctiva with Rickettsias, these being the active principle of the trachomatous virus.

"We consider that we have proved both morphologically and experimentally that trachoma is a Rickettsiosis, and that the louse forms a reservoir, and is perhaps one of the vectors of trachomatous virus, probably the principal one."

W. ZENTMAYER.

THIRD NOTE ON THE BACTERIAL AGENT OF TRACHOMA. A. CUÉNOD and R. NATAF, Arch. d'opht. **53:** 218 (March) 1936.

In this report Cuénod and Nataf consider the pathologico-anatomic features as shown by sections of trachomatous tissue which corroborate and clarify the clinical observation made by the use of smears. They have observed a powdery coccobacillary figure filling the spaces between the hypertrophied nodes of epithelioid cells which can hardly help being thought of as the multiplication of a parasitic agent of lower virulence which is of such a nature as to account for the pathologic hyperplasia of the nodes as well as the cytolysis of the peripheral cytoplasm of the epithelioid cells. Consideration of these cells suggests the idea of epitheliosis. Some support is gained from the latest work of Morax and especially the work of Thygeson.

S. B. MARLOW.

THE PATHOLOGY OF TRACHOMA. R. P. WILSON, Bull. Ophth. Soc. Egypt **29:** 1, 1936.

The characteristic infiltration in trachoma is composed of lymphocytes, mononuclear leukocytes, plasma cells and histiocytes. Polymorphonuclear leukocytes are present only in mixed infection. Since the expressible follicles of the second stage of trachoma are found chiefly in the retrotarsal folds, they cannot be the sequelae of strangulation of the meibomian ducts, as no meibomian ducts are found in this situation.

J. E. LEBENSOHN.

Tumors

A CASE OF DERMO-EPITHELIOMA OF PARINAUD. G. RENARD, R. HUGUENIN and P. CASSIAU, Arch. d'opht. **53:** 197 (March) 1936.

In 1884 Parinaud described a tumor "reddish yellow in color, semitranslucent, sometimes lobulated, situated outside the margin of the cornea, movable on the sclera, and occurring in young subjects," to which he gave the name dermo-epithelioma. The authors have collected fifty-two instances in which there was an essentially similar histologic picture but in which the tumor was described under varying designations. They report in detail the results of observation of a man 67 years old

who ten years before had had subconjunctival injections and other treatment for detachment of the retina. He had seen no physician for seven years prior to observation. A tumor was present in the conjunctiva near the caruncle, which after removal was revealed by histologic study to be a dermo-epithelioma closely resembling that described by Parinaud. As a result of the study of all the observations reported the following facts are noteworthy: 1. The tumor is almost always situated in an exposed portion of the conjunctiva. 2. In three cases it has been associated with a pterygium. 3. In four cases there has been evidence of trauma to the conjunctiva at the site of its occurrence. While the term dermo-epithelioma is not perfect, the authors believe it to be better than any other so far proposed. The diagnosis is simple; there is no conjunctival irritation or pain. Excision usually results in complete cure.

S. B. MARLOW.

A CASE OF LYMPHOCYTOMA OF THE ORBIT. M. DE TREIGNY, Bull. Soc. d'opht. de Paris, June 1936, p. 406.

A patient aged 56 years had exophthalmos of the right eye of three years' duration. Surgical intervention had been instituted one year previously. The proptosis was direct, nonpulsating and nonreducible and gave the impression that a mass was behind the globe. Visual acuity was 0.7, and the fundus was entirely normal. During a year's observation the exophthalmos was slightly increased, and on palpation a firm lobule presented itself at the inferior external angle of the orbit. The conjunctival vessels were greatly dilated; the globe was only slightly movable, and the center of the cornea was ulcerated and stained. A roentgenogram showed a soft shadow of the entire orbit and the sphenoidal opening. Operation performed through an orbital incision at the external canthus for the purpose of removing a specimen for biopsy failed to alter the exophthalmos. Histologic examination revealed a homogeneous mass of lymphoid tissue with some large fibrous strands running through it. The cells were lymphocytes with a great number of mitotic figures and some large cells with much clear reticulum. Application of radium on the external portion of the orbit was instituted. Ten applications were given; after the second amelioration began, and there was complete reduction of the exophthalmos at the end of the course of treatments. Slight limitation of the movements of the globe persists. General examination gave no positive findings. The observation illustrates the relatively benign nature of these tumors and their extreme radiosensitivity.

L. L. MAYER.

PREOPERATIVE AND POSTOPERATIVE OCULAR SYMPTOMS OF TUMORS OF RATHKE'S POUCH. F. SPINELLI, Riv. oto-neuro-oftal. **13**: 489 (Nov.-Dec.) 1936.

Spinelli describes eight cases of Rathke's cysts before and after surgical intervention. The purpose of this study was to establish whether the ocular findings could furnish enough data to enable one to localize the tumor. The majority of modern authors are of the

opinion that tumors of Rathke's pouch develop between the anterior and the posterior lobe of the hypophysis. They can therefore cause pressure on the hypophysis, on the optic chiasm and on the optic pathways. If they develop more extensively they may damage the third ventricle, the cavernous sinus, the cerebral peduncles or even the internal carotid artery.

In Spinelli's eight cases operation was performed by the frontotemporoparietal method, and Rathke's cysts were found varying in size from that of a small nut to that of a chicken's egg. In almost every case the cyst was adherent to one or both optic nerves, causing pressure on them. Increased intracranial pressure was noted in two cases, headaches in five cases, vomiting in three, vertigo in one and polyuria in one. Differences in vision in the two eyes were noted. In two cases vision was 10/10 in each eye. However, in the others there was a definite difference between the two eyes. In one case vision was 20/20 in one eye and in the other was reduced to perception of movements of the hand. The visual field varied. In seven cases it was affected bilaterally; in one case the one eye was normal, while in the other there was only residual vision in the inferonasal quadrant. Classic bitemporal hemianopia was found in only one case; in another case there were absolute temporal hemianopia in the right eye and relative temporal hemianopia for white and total achromatopsia associated with a relative central scotoma for white in the left eye. The fourth patient presented concentric contraction of the fields. The fundi showed either temporal or descending atrophy of the optic nerve. In one case there was papillary edema. The roentgen rays showed constantly lesions of the sella turcica, especially in the posterior portion. It is noteworthy that the age of these patients ranged from 6 to 29 years.

Spinelli concludes that while examination of the fundus, accurate determination of the visual fields and screen tests are of importance, one must also make an accurate study of the x-ray plates so as to localize the tumor and proceed to surgical treatment.

In every case, after removal of the cyst the atrophic change in the optic nerve was arrested.

G. BONACCOLTO.

Tumors

PROGNOSIS OF SARCOMA OF THE UVEA. H. DENECKE, Klin. Monatsbl. f. Augenh. **97:** 594 (Nov.) 1936.

Denecke refers to previously published observations on the prognosis of uveal sarcoma, with which his own observations coincide. These observations were made on thirty-six patients treated at the University Eye Clinic of Greifswald within the last twenty years. Their ages ranged between 40 and over 70 years, the decade of 51 to 60 years being chiefly represented. Sarcoma of the choroid predominated, being present in twenty-six patients; the ciliary body was afflicted in four and the iris in two. Sarcoma of the ciliary body showed relatively the largest number of metastases, namely three in four cases. Fourteen patients died from metastases, eleven of these within five years after enucleation, and one of them fourteen and one-half years after this operation.

Denecke considers the first five years after enucleation as the critical period, which should be considered in writing life insurance policies. The risk is small after the lapse of ten years.

Pigmented sarcoma showed no greater tendency toward metastases than the nonpigmented type; in nineteen of the thirty-six cases the tumor was pigmented. Exenteration of the orbit was performed in seven cases; in such cases the prognosis is not absolutely unfavorable, as one of these patients was still living after twelve and one-fourth years. In one patient perforation into the orbit was histologically noted after enucleation; he was living thirteen years afterward, although no exenteration of the orbit was performed. Local recurrences were not observed.

K. L. STOLL.

Visual Tracts and Fields

ANGIOSCOTOMAS. H. VIALLEFONT and R. LAFON, Arch. Soc. d. sc. méd. et biol. de Montpellier **17:** 122, 1936.

Using each other as subjects, the authors plotted the angioscotomas before and after inhaling amyl nitrate. No change was noted.

J. E. LEBENSOHN.

Visual Tracts and Fields

A CASE OF DOUBLE HEMIANOPIA. J. MALBRÁN and E. DE LA RIEGA, Arch. de oftal. de Buenos Aires **11:** 538 (Sept.) 1936.

The authors report a case of double hemianopia with preservation of the macular region and a quadrant below. The literature is extensively reviewed, and attention is called to a similar condition reported by Wilbrand and Sachs and classified under type XXI in the classic work of Wilbrand and Sänger.

C. E. FINLAY.

Therapeutics

UTILIZATION OF SODIUM HYPOSULFITE IN OCULAR THERAPEUTICS. A. DUBOIS-POULSEN, Bull. Soc. d'opht. de Paris, July 1936, p. 560.

Sodium hyposulfite has been used for a long time in general therapeusis. In 1860 Polli experimented with its protective properties. The revival of its use was inaugurated by the work of Ravaut in 1920. In intolerance, anaphylaxis and allergic states it has been of much value as a desensitizer. Its use for eczema is well established. Dubois-Poulsen has found the drug efficacious in eczema of the lids, blepharitis, certain forms of conjunctivitis and certain forms of keratitis. He lists a group of patients who have benefited by this therapy. No untoward results or complication were observed. A review of the pharmacodynamics of sodium hyposulfite is given.

L. L. MAYER.

VALUE OF ATROPINE IN CASES OF TABETIC ATROPHY. J. FEJER, Arch. f. Augenh. **110:** 76, 1936.

No improvement in the visual acuity could be found in cases of tabetic atrophy of the optic nerve following retrobulbar injections of atropine, as advised by Zamkovsky and by Springovitsch (*Klin. Monatsbl. f. Augenh.* **90:** 343, 1933). In those patients who were followed over a long period it seemed to Fejer that the atropine had some beneficial effect in staying the disease process. Favorable results were observed in atrophy of the optic nerve following retrobulbar neuritis when this method of treatment was used.

F. H. ADLER.

Society Transactions

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Annual Congress, London, England, April 29 and 30 and May 1

DR. GORDON HOLMES, C.M.G., C.B.E., F.R.S., *President*

THE PROGNOSIS IN PAPILLEDEMA. DR. GORDON HOLMES.

Papilledema is now recognized to be essentially edema of the nerve head, in the early stages of which there is little structural damage or functional disturbance of the nerve elements, which later may be strangled and destroyed by overgrowth of interstitial tissue. In the latter stage, recovery of vision is not to be expected. The essential etiologic factor in papilledema is increased intracranial pressure. Relief of that pressure, if effected before the development of secondary changes in the disk, will lead to disappearance of the condition and so will remove the risk of blindness. In rare cases the papilledema subsides without surgical intervention or other specific treatment, and vision remains unaffected.

In most cases of papilledema, relief of intracranial pressure becomes necessary in order to save sight. The more rapidly the congestion and swelling of the optic disk develop, the more intense becomes the papilledema and the greater the danger to sight if relief is not undertaken. Swelling of 4 or 5 D. is a warning against undue delay. Equally serious are great engorgement of retinal veins and early and extensive hemorrhages on the surface of the swollen disk. An even more important danger is narrowing of the arteries in the swollen disk and its vicinity. Frequent examination should be made in order to evaluate any changes occurring in the fundus and to detect the earliest signs of atrophy of the optic nerve. A further serious sign is the transient loss of sight, which may occur after sudden change of posture. If the tumor or other cause of the increased pressure cannot be removed, decompression craniectomy usually causes subsidence of the congestion in from two to four weeks, and if secondary changes have not appeared by then there is no further risk of impairment of vision.

RETINAL CIRCULATION: CHANGES IN METABOLIC DISEASE. MR. A. E. MACDONALD, Toronto, Canada.

From the material presented the speaker drew the following conclusions: 1. Papilledema which is indistinguishable from that in cases of tumor of the brain occurs in some cases of metabolic disease. 2. In the latter the eye is subject to injury by toxins in equal degree as other parts, but the outstanding retinal changes cannot be explained by toxins, arteriosclerosis or inflammation. 3. Obstruction of the ocular drainage develops with an explosive, a slow or an intermediate time factor. 4. Venous distention at the disk occurs late in systole and disappears when the intracranial pressure is raised to about 250 mm.

of water or 18 mm. of mercury. 5. Straining, lifting, coughing and holding the breath produce elevation of the intracranial pressure sufficient to cause obstruction of the central vein.

CATHOLYSIS IN THE TREATMENT OF RETINAL DETACHMENT. MR. R. FOSTER MOORE.

The object of catholysis is to produce coagulation in the choroid as the result of the liberation of sodium ions at the negative terminal inserted through the sclerotic. At the same time hydroxyl ions are set free and can be readily seen with the ophthalmoscope during the operation. The platinum iridium needles which I use are either straight or bent at a right angle. The length of the active part of the needle varies from 1 mm. upward. The needles can easily be passed through the muscles into the globe, and when used thus must be from 4 to 5 mm. in length. The optimum strength of current is 5 milliamperes, applied for six seconds. An almost unlimited number of punctures can be made, even though the eye may be somewhat soft. Bubbles of gas are seen at the site of the puncture, and from the punctures there is always some escape of fluid. Frequently no additional exit is required for the outflow of subretinal fluid.

Of 31 patients treated by this method, 14 were myopic. In 12 the retina was replaced, and was in position when the patient was last seen. Two patients were discharged slightly improved, 4 much improved and 9 not improved. The longest posttreatment period has not been over twelve months and in many of the cases has been only a few weeks. The advantages of the method are that the apparatus required is light, portable and easily used, and the effects of the procedure are sharply localized, with less damage to intra-ocular tissues than results from diathermy. It is unnecessary to divide the muscles unless exposure of the sclerotic far back is required. The crucial test for success is not in how many cases the retina goes back but in what proportion the replacement is permanent.

CRITICISM AND COMMENT ON NEWER METHODS OF TREATING DETACHMENT OF THE RETINA. MR. J. COLE MARSHALL.

In catholysis the whole length of the needle is active, whereas in diathermy the only active part of the needle is its base, and often the point becomes cool. In catholysis, keratitis and retinitis are formed beyond the region where the bubbles have been liberated, so that when the retina goes back the retinitis in that region may cause changes in the choroid. Mr. Foster Moore retains the needle in the eye longer and uses a slightly stronger current than do most continental operators. Certainly catholysis is a good localizing agent, and it is also a good adjunct to diathermy scarring. In cases of detached retina I have found catholysis very beneficial when used in conjunction with diathermy, and for the latter the use of very fine needles represents a distinct advance. The transillumination method described by Weve I regard as very important, as is also the continuous use of saline solution during the operation.

EXOPHTHALMIC OPHTHALMOPLEGIA. DR. W. RUSSELL BRAIN.

The syndrome of exophthalmos and ophthalmoplegia may occur either unilaterally or bilaterally. When arising spontaneously it is associated with general symptoms of thyrotoxicosis. But it may follow thyroidectomy for hyperthyroidism, in which case general symptoms of thyrotoxicosis are often absent, and the basal metabolic rate is normal or even subnormal.

The separation of this syndrome from exophthalmic goiter depends on a number of features, the most important being that it may occur postoperatively, not only in the absence of thyrotoxicosis but in the presence of actual hypothyroidism. It also differs from exophthalmic goiter in its age incidence and its sex incidence, in the usually slight degree of the thyrotoxic symptoms, when these are present, in the lack of response to thyroidectomy and in the somewhat atypical histologic picture presented by the thyroid. As to the period of life at which cases are observed, exophthalmic ophthalmoplegia is a disorder of middle age. The mode of onset is usually subacute: One eye becomes increasingly prominent over a period of from three to four months, ophthalmoplegia and double vision developing concurrently. Generally the other eye lags behind in the matter of proptosis and may not exhibit ophthalmoplegia. Both exophthalmos and ophthalmoplegia may develop simultaneously in each eye in three or four months. Exophthalmos was present in all my cases; it was unilateral in 5 and bilateral in 24. In the latter cases the degree of exophthalmos of the two eyes was equal in 8 instances and unequal in 16. Asymmetry of the proptosis was present in 21 of 29 cases. Ophthalmoplegia was unilateral in 12 of the cases and bilateral in 17. The ophthalmoplegia was paresis or paralysis affecting not movements of individual extra-ocular muscles but movements of the eye in a particular plane. Among the cases in which the ophthalmoplegia was unilateral, elevation was the movement most often affected; in the cases in which it was bilateral, abduction was affected in 31, elevation in 23, depression in 18 and adduction in 17. In 6 patients all movements of each eye were affected.

Widening of the palpebral fissures associated with retraction of the upper lid was the usual finding in both cases of unilateral and those of bilateral ophthalmoplegia, but proptosis was present on each side in 5 cases and on one side in 3 cases of bilateral ophthalmoplegia.

With regard to the pathologic changes of the ocular muscles and the thyroid, these consisted of marked edema, with foci of lymphocytic infiltration and, in later stages, fibrosis. In one portion of the levator palpebrae superioris muscle, which was removed at operation, Professor Turnbull noted general edema and great enlargement of the muscle fibers. In 4 of 5 cases, sections of the thyroid showed that the gland was atypical, and in the other case the gland was in the same condition as in cases of exophthalmic goiter.

In my experience, the usual treatment for exophthalmic goiter has been disappointing when applied for exophthalmic ophthalmoplegia. Thyroidectomy was done in 4 cases. In 1 of these cases there were marked general improvement, and considerable improvement in the condition of the eyes, though some ophthalmoplegia remained. After

orbital decompression immediate recession of the eye occurred, but the ophthalmoplegia remained unchanged in 1 case and was only moderately benefited in the other 2. I hope the day will come when the condition can be dealt with physiologically.

PROF. F. R. FRASER: I have been able to test the effect of prostigmin on 2 patients with exophthalmic ophthalmoplegia. During the last twenty-five years there has been a great advance in the knowledge concerning the transmission of nerve impulses to muscles and especially as to the importance of acetylcholine. This substance, it has been shown, is essential for the transmission of nerve impulses across the ganglions of the sympathetic system, for the transmission of impulses from the vagus nerve to the effector organs of the vagus system and also for the transmission of nerve impulses from nerve endings to voluntary muscles. But the substance is rapidly destroyed in the body; otherwise, the muscle would remain in a state of tetanic contraction. It is destroyed in the presence of choline esterase.

It has frequently been noted that there are similarities between toxic goiter and myasthenia gravis. Many persons who suffer from the former disease complain of severe muscular weakness and excessive fatigability; the similarity is most marked in patients with goiter in whom exophthalmic ophthalmoplegia develops. In both cases of toxic goiter and those of myasthenia gravis, lesions of the thymus gland have been recorded, and on histologic examination the muscles, in both cases of myasthenia gravis and those of ophthalmoplegia, show collections of lymphocytes. Dr. Mary Walker has recently shown that a synthetic analog of physostigmine known as prostigmin restores voluntary muscle in cases of myasthenia gravis. The effect is dramatic, as it is evident a few minutes after this drug has been subcutaneously injected. In a few hours the effect passes off. This action is due to the inhibition by prostigmin of the esterase. No abnormality in the choline esterase of the serum was present in myasthenia gravis, and a disturbance of the normal balance between the production of acetylcholine and the activity of the esterase at the neuromuscular junction is supposed to exist.

The first patient with exophthalmic ophthalmoplegia on whom prostigmin was tried was a woman aged 41 who at the age of 26 had begun to show signs of toxic goiter; those evidences were tremors, palpitation and marked sweating. A year later she showed exophthalmos, which was more marked on the right than on the left, diplopia and pronounced ptosis of the left upper eyelid. The ophthalmoplegia developed rapidly and interfered with her work, which was teaching school. She had at this stage drooping of the eyelid, which became more marked toward the end of the day, and at the end of the school term the diplopia was much more severe. In 1925 partial thyroidectomy was carried out, and after this the ophthalmoplegia became much better, and the ptosis on the left was so much improved that she was able to resume her teaching. The tachycardia and tremulousness, however, never disappeared. She was, therefore, treated with prostigmin, 1 mg. of atropine having first been used as a preliminary medicament. Twenty-three minutes after administration of the atropine, 2.5 mg. of prostigmin was given, a large dose, the possible upsetting effects of which were

counteracted by the atropine. Four minutes after the prostigmin was given there was improvement in the upward vertical movement of the right eye; in a few minutes the axes of the eyeballs became parallel when the patient looked straight forward; there was twitching of the orbicularis muscle, and diplopia was present only when she looked above the horizontal level. Seventeen minutes after the injection, diplopia was to be found only on extreme upward elevation of the eyes. At that stage the patient's condition began to retrogress a little, as shown by the fact that diplopia could be elicited more easily, and after half an hour the axes were again deviated. She said that during the rest of the evening her eyes were stronger. A week later a similar dramatic result was brought about in the same manner.

The second patient was also a woman aged 41, who had had toxic goiter when she was 32, the onset of which was marked by lack of energy and a feeling of weariness. A year later, exophthalmos was noticed, but there was absence of severe toxicity. Then diplopia appeared. Thyroidectomy was done in 1930, and since then the ophthalmoplegia has remained stationary. During the last few years the patient has been treated with thyroid because of her feeling of lassitude and general depression, and in the last year the exophthalmos has increased to a greater degree than at any time before. Prostigmin was used, but in this patient it had no effect.

The difference as to the result of treatment with prostigmin in these two cases suggests a different condition in them, or it means that the disease process in the second patient had progressed to such an extent that it could not respond to the drug.

A Case of Exophthalmic Ophthalmoplegia. Mr. H. B. Stallard.

A man aged 31 complained that on looking upward and on opening the eyes after they had been closed two or three minutes he had diplopia. His condition was becoming progressively worse. He had a tired feeling in his eyes and general lassitude; he was also nervous and easily worried. There was evident bilateral exophthalmos, which was more marked on the left. The thyroid was slightly enlarged, soft and of uniform consistence. The pulse rate varied from 64 to 90, the reflexes were brisk, and the basal metabolic rate was —40 per cent. No abnormality was found in the blood count or from a roentgenogram of the skull. The Wassermann reaction was negative. The pharyngeal lymphoid tissue was reported to be inflamed. Injection of 600 organon units of the thyrotropic hormone of the anterior lobe of the pituitary was given on three successive days, to permit study of the effect of that substance on the activity of the thyroid and the basal metabolic rate. The latter rose from 3 to 21 per cent within twenty-four hours after the first injection was given; a further rise to 30 per cent took place after the second and third injections, and in three days the rate rose to 41 per cent. During the twelve hours after the first injection the temperature rose, and there was a corresponding increase in the pulse and respiration rates. Frontal headaches occurred ten hours after the first injection and eleven hours after the second and third. Then the patient slept; the next morning the headache was only slight, and it did

not occur again. Three weeks later a corneal ulcer developed in the left eye, and in spite of tarsorrhaphy, phenolization and paracentesis this eye lost its sight.

DISCUSSION

DR. GORDON HOLMES: In a certain proportion of the cases of proptosis of the eyes which I have observed there have been few other symptoms of thyrotoxicosis, and, in particular, there was no increase in the pulse rate. It is possible that there are three types of this condition. Professor Fraser's results in 2 cases indicate two types, and I have seen instances of a rarer form, one characterized by definite weakness of some of the ocular muscles, with rapidly developing exophthalmos. In reports of cases of thyrotoxicosis some subluxation of the globe has been described. I think the transient palsies are caused by sudden stretching of the muscles, due to mechanical causes, in the orbit itself. As to the pathologic basis, Sir William Gowers, many years ago, mentioned the possibility of primary degeneration of the cells occurring in these cases. Vigor demonstrated leukocytic infiltrations in myasthenia gravis, but Mr. Stallard has now reported that in his case the muscle fibers did not show pathologic change. The question whether there is in this condition a change in the nerve supplies of the muscles has not been answered. In exophthalmic ophthalmoplegia there is some variability in the symptoms; several of my patients have said that their ocular movements were fuller in the morning than at the end of a tiring day. This exhaustibility of ocular movements brings this condition more into line with myasthenia gravis, a point which Professor Fraser has mentioned.

DR. RUSSELL BRAIN: In 1 case serial sections were made of the brain stem, and no abnormality was found in the nervous system. Some ophthalmologists advocate bandaging the eye, but I always found that this made the condition worse.

PROF. F. R. FRASER: Prostigmin reaches its full effect in half an hour after injection, and the effect remains obvious for four hours. When the method of injection is used, the drug has to be given every four or five hours. Less than 2.5 mg. is not of much use in myasthenia gravis. A much smoother action is obtained when the drug is taken by mouth, but with this method of administration large doses must be taken.

SPONTANEOUS CURE OF RETINAL GLIOMA. MR. MONTAGUE L. HINE.

Paintings were shown of three eyes in which nature had evidently cured early gliomatous growths. In the last edition of his textbook Sir John Parsons gave the prognosis of glioma of the retina as being wholly poor, the patient invariably dying of the condition if untreated. In 1933 Griffith recorded particulars of a "glioma family," and a fuller history was given in his report. The father, aged 42, had his left eye excised in infancy because of retinal glioma, and on recent examination his other eye showed on the nasal side of the fundus an atrophic area of the retina and choroid on which was superimposed a shrunken, slightly raised cystic mass of retina, which was presumably, if the history is borne in mind, a regressed and cured patch of old glioma. The

eldest child died at 6 months of age; the second child, now 19 years old, was found to have cystic masses in each retina, that in the right eye being typical of previously described spontaneously cured retinal glioma. The third child died of retinal glioma, which was bilateral, at 21 months of age; the fourth had one eye excised for the same condition when 3 months old, and the fifth died of bilateral glioma at 4 years of age.

DISCUSSION

SIR JOHN PARSONS: I consider that my general statement in my textbook for students on this matter is correct. I am very doubtful about recorded cases of spontaneous cure of this condition, and, in any case, it would be wrong and dangerous to lead students to expect nature spontaneously to cure this serious condition. Every case of so-called cure should be backed up by report of histologic examination by experts.

MR. R. FOSTER MOORE: I have observed a similar case of spontaneous cure. I think there can be no reasonable doubt about these instances of cure which have been reported.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

May 17, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

CHANCRE OF THE UPPER EYELID IN AN INFANT TWO MONTHS OF AGE. DR. ALFRED APPELBAUM.

An apparently normal infant of 8 weeks showed on the right upper lid a sharply defined oval ulcer, with its longest diameter in the horizontal direction, 3 mm. high, mounted on a painless induration. The surface was level, dry, crustlike, and 0.5 mm. below the level of the border of the slightly edematous encircling skin. The corresponding preauricular glands were enlarged. The initial dark field examination gave negative results, but three days later, examination disclosed Spirochaeta pallida. The source of infection was late secondary syphilis, which appeared in the mother one month after delivery. She had had antisyphilitic treatment four months before pregnancy commenced, discontinuing the therapy shortly thereafter. The Wassermann reaction of the blood was negative just before and after midterm and at delivery.

SYNCHYSIS SCINTILLANS IN THE ANTERIOR CHAMBER. DR. GIROLAMO BONACCOLTO.

Synchysis scintillans in the anterior chamber was seen in a woman 64 years of age. Examination showed the right eye to be congested and hypertonic. Under the slit lamp myriads of spherical bodies of various dimensions were seen, from which many colored lights were reflected.

Other writers have reported these bodies as polygonal crystals. Seen with the naked eye they appear as gold flakes or crystals in the aqueous or the vitreous, but under the slit lamp they have a spherical appearance.

DISCUSSION

DR. WENDELL L. HUGHES: This reminds me of a case that my associates and I observed at the New York Eye and Ear Infirmary six months ago, in which the anterior chamber was entirely filled with crystals and secondary glaucoma developed later. We did a paracentesis, collecting the fluids that were withdrawn, and under the microscope these were shown to be cholesterol crystals. The glaucoma was controlled by removal of the material from the anterior chamber. This was apparently degenerated vitreous.

PAGET'S DISEASE OF THE SKULL. DR. W. GUERNSEY FREY.

A 49 year old man with Paget's disease of the skull (osteitis deformans) presented the following ophthalmologic findings: alternating squint, papillitis in a stage of beginning atrophy, contracted fields and enlarged blindspots. There was no evidence of changes at the orbital apexes on roentgen examination.

DISCUSSION

DR. MARTIN COHEN: I should like to ask whether the ocular changes are due to Paget's disease or whether the ocular manifestations might not be due to some vascular disturbance having no bearing on Paget's disease. In a number of cases of Paget's disease the fundi have been absolutely normal, and I reported a case in a man 70 years of age who had lesions of the fundus similar to those shown by Dr. Frey's patient this evening. Autopsy in that case showed the characteristic bony changes in the skull. The vascular changes in the eyes were attributed to marked capillary sclerosis of all the choroidal vessels, and this condition might explain the capillary hemorrhages which occurred in the fundus of one eye in my patient.

DR. JOHN H. DUNNINGTON: I have followed a woman with Paget's disease for many years. She has vision of 4/200 in one eye; the other eye has no perception of light. The changes in the fundus are not typical. There is some atrophy of the optic nerve, but it seems to me that the loss of vision is out of proportion to the atrophy of the optic nerve. A younger woman, who has very advanced Paget's disease, has retained vision of approximately 20/40 in each eye. She has peripheral contracture of her fields of 10 degrees and a certain amount of atrophy of the optic nerve. There are no vascular changes in the fundus of the younger patient, but there is marked sclerosis of the choroidal vessels in the older woman.

DR. W. GUERNSEY FREY: We should all like to know what causes the ophthalmologic symptoms in Paget's disease. I have talked to a number of neurologists, and they are largely of the opinion that the neurologic symptoms are due to pressure on the nerves. I am hardly convinced of that myself after what I have read in the literature. It appears to me, since it is not known what the cause of the

condition is in the first place and since the bony lesions seem to be trophic disturbances, that the same underlying condition may be responsible for the ocular signs.

LAURENCE-MOON-BIEDEL SYNDROME. DR. H. TEMPLETON SMITH.

It is of interest to ophthalmologists that although only one of the five cardinal manifestations of the Laurence-Moon-Biedel syndrome is ocular, the first cases to be recognized and reported were observed by ophthalmologists.

Since Laurence and Moon first reported this syndrome in 1866, only about 80 cases have been reported. The characteristic findings are: (1) polydactylism; (2) obesity, which comes on after a few years of age, with the general appearance of pituitary imbalance; (3) genital abnormality of varying degrees; (4) a condition ranging from mental deficiency to imbecility; (5) atypical retinitis pigmentosa, and (6) frequently a familial tendency.

The etiologic factors have been lightly touched on by the observers. The first and most stressed factor is the hypophysis. In the series, the results of the basal metabolic test in 17 cases were reported. In 2 cases the basal metabolic rate averaged + 25; in 4 it was normal; in 4 it was low, and in 7 it averaged — 25. A survey of the literature reveals only one autopsy, with no abnormal findings except those grossly observed in the living patient.

The case of a Puerto Rican boy 14 years of age, the third child of a normal mother, was reported. Two brothers were normal. The patient had enjoyed normal health and vision up to 5 years of age. The first sign of any abnormality was failure of vision, followed by general sluggishness and slowing of all reactions.

Examination showed a supernumerary maldeveloped finger on each hand, an unhealthy pallor, small hands and feet with no muscular strength, and obesity. The Wassermann and Kline reactions of the blood were negative, and the blood chemistry was normal. The hemoglobin content was 63 per cent, the red cell count 4,700,000 and the white cell count 8,700. The basal metabolic rate was — 18. A roentgenogram showed the sella turcica to be moderate in size, without erosions, and the clinoid processes did not encroach on the pituitary space. Owing to lack of intelligent cooperation of the patient, the amount of vision and the fields could not be determined. In attempting to cross the room with various obstacles in his path the patient would bump into each one. External examination of the eyes gave negative results; the pupils were equal and reacted to light. The state of accommodation was not determined. The media were clear, and the optic nerves were oval and had a slight waxy pallor. The lumens of the vessels were contracted and slightly irregular. Scattered throughout both retinas were areas of atypical patches of retinitis pigmentosa, with some degeneration of the retina.

DISCUSSION

DR. JOHN M. MCKINNEY: I saw this patient with Dr. Smith at St. Luke's Hospital. The parents of such patients as a rule do not pay much attention to the polydactylism; the first thing that attracts

their attention is the poor vision, so the ophthalmologist is often the first physician to see these patients. That was so in the first cases of this syndrome to be described in 1866 by Laurence and Moon, the British ophthalmologists. I was interested in the fact that the famous English physician Jonathan Hutchinson also examined these patients. The original report was buried in the literature until 1922; it was dug out by Biedl, the eminent Czechoslovakian endocrinologist, and that is why his name is added to the syndrome. There has been a great deal of talk about the treatment. In some cases the patient has responded to thyroid and pituitary therapy, but such treatment is apparently empirically used, for, as far as can be made out, the condition is not glandular in etiology but is probably a congenital defect on the basis of a defect in the germ plasm. This is borne out by the fact that in the majority of cases the disease is familial; that is, one or more members of the same generation are affected; but the syndrome is not hereditary.

In the interest of differential diagnosis, the patients are nearly always children when first seen, and if there is retinitis pigmentosa associated with striking obesity of the hypopituitary type, small genitalia, polydactylism, mental deficiency and familial incidence, the case falls into this diagnostic group.

DR. HARRY V. JUDGE: Appreciating the rarity of this syndrome, I desire to mention a case observed about three years ago. A child aged 4, the first-born of Jewish parents who were second cousins, was brought to me on account of convergent strabismus of the left eye. The child presented in the main the symptoms outlined by Dr. Smith, namely, marked mental deficiency, polydactylism, syndactylism, obesity, undescended testicles and a rudimentary penis. There were an extra digit on the right hand and an extra digit on each foot. The mental state precluded determination of the visual acuity. There was a fine lateral nystagmus with an occasional vertical component. On refraction the retinoscopic test showed need of a —7.50 D. sphere for the right eye and of a —9.0 D. sphere for the left. The fundus did not present the usual atypical retinitis pigmentosa as seen in the patient exhibited tonight, but the retina had the appearance of one lacking normal structure and showing tessillation with deeply pigmented islands between the vessels of the choroid—a picture somewhat similar to that in the case reported by Savin (*Brit. J. Ophth.* **19:** 597 [Nov.] 1935) but without the pigmentary changes noted in that case. It is possible that pigmentary changes may take place at a later date. The disk was dirty yellow and showed a sharp outline and contracted vessels. It is possible that central pigmentary changes were present, but, owing to the nystagmus and the poor cooperation of the patient, it is impossible to say with certainty. Two years ago an attempt was made by surgical intervention to bring the testicles within the scrotum, but the operation was unsuccessful. Prior to this, an extract of urine of pregnant women had been administered but resulted only in development of pubic hair.

The pictures show the characteristic stigmas. The members may have difficulty in observing the bilateral inguinal scars.

At the present time at the Albany Medical College some work is being done by Dr. James Hamilton, the endocrinologist, with an androgen known as testosterone propionate (used by Ruzicka, of Basel,

Switzerland) in cases of cryptorchidism. His results in studies on animals have been startling, and in the case of one human being he has reported a decided improvement in this condition.

My associates and I are anxious to have this drug tried in this case of the Laurence-Moon-Biedl syndrome to ascertain the effects on the genitalia, but we believe the action of the drug will be somewhat inhibited on account of the previous surgical intervention. The second boy in this family also shows cryptorchidism, but a cursory examination of his fundus showed none of the signs present in his brother's. The cryptorchidism may be an expression of familial hypopituitarism.

RELATION OF AMETROPIA TO OCULOMOTOR ANOMALIES. DR. JAMES W. WHITE.

The many exceptions to the general rules for the correction of ametropia, due to anomalies of the ocular muscles and accommodation, have not been stated. The reasons for the need of glasses were stated, and it was explained why ametropia may be better uncorrected, overcorrected or undercorrected, depending on the result of the tests and the presence or absence of symptoms.

DISCUSSION

DR. JOSEPH I. PASCAL: Dr. White advised correcting myopia fully when it is associated with exophoria. This is the old and standard teaching, and such correction is unquestionably feasible in a great many cases. It is predicated on the theory that the myopia is primary and the exophoria is secondary. But there is another side to this problem in cases in which the relationship seems reversed, that is, in which the exophoria is primary and the myopia secondary. Correcting the myopia fully in these cases without first treating the basic cause, i. e., the exophoria, would probably not give the best results. In early childhood, especially, it seems that such exophoria brings about functional overactivity of the associated accommodation, producing at first functional myopia, which in time becomes real myopia. In such cases correcting the myopia fully seems to work contrary to the principles suggested by the etiology and may lead to a further increase of myopia, whereas relaxing the overconvergence by correcting the exophoria with a prism base in or by some dissociating exercises seems to work more in harmony with the principles suggested by the etiology. It is also a fact that correcting the myopia fully generally does not lessen the amount of associated exophoria, which would also speak against the myopia being primary. Cases of primary exophoria with secondary myopia do occur, perhaps more often than is believed, and I should like to ask Dr. White what he thinks of these cases, drawing on his wide experience.

DR. JOHN H. DUNNINGTON: I am thoroughly in accord with everything Dr. White has said, particularly the fact that there are many myopes who do not care to wear glasses. I am a myope and prefer to wear my glasses and see, but there are many people who do not. I cannot understand why the ophthalmologist should insist on the use of glasses when the patient does not wish to wear them, unless the condition has some pathologic bearing on a muscular anomaly. It has been my experience that moderate myopia does not increase because

of failure to wear the full correction, nor does exophoria become worse under these conditions.

DR. JAMES W. WHITE: In regard to theories of exophoria and myopia, I shall refer to my first statement or two, that rules and theories are all right if they work. If one finds an instance in which exophoria seems to be primary and myopia secondary, all right. If one finds an instance in which myopia is primary and exophoria secondary, all right, too; one should get proof of it. But I emphasize again one of my first statements: One should examine the patient before giving the cycloplegic, and then after, and as many times after that as is thought necessary. One should examine the patient under different conditions and see how the amount of deviation varies. Granted that one will find patients with primary exophoria who have myopia, I also know that one will find a good many patients with myopia in whom exophoria develops.

OPERATIVE INJURIES OF THE EYE. DR. PERCY FRIDENBERG.

Operative injuries of the eye occur not only in ophthalmic surgical procedures, but in operations on the accessory sinuses, intranasal instrumentation and obstetric procedures. All forms of traumatism, from corneal abrasion to laceration of the optic nerve, and material and personal hazards in ophthalmic operations were noted.

DISCUSSION

DR. MARTIN COHEN: The one point which struck me during the presentation of the paper was the nonuse of the speculum. It seems to me that operative procedures on the muscles, or extra-ocular procedures, require a speculum. I saw in Paris a procedure for extraction of cataract by Frouseau in which he separated the lids with the thumb and forefinger, introduced the cataract knife in the usual way, did a capsulotomy and then gently expressed the lens. He used no speculum and applied his thumb on the cornea to express the lens. I have seen him perform that procedure frequently, without any ill effects on the globe, which shows the importance of avoiding the use of the speculum in operations on the globe as much as possible.

DR. ERNST WALDSTEIN: Czermak in his later period used a speculum, and the assistant held the lids with his fingers only in exceptional cases. Elschnig utilized his assistants' fingers for holding the lids very sparingly; his first intracapsular extractions after Smith's method were performed with this precautionary measure. For my part, I have always doubted its advantage compared with the use of the speculum, especially when one keeps the latter unlocked, as Elschnig always urged. In order to get a good hold of the lids the fingers have to grasp them close to the edge, and then it is often almost next to impossible to avoid any undue pressure, the very thing one tries to prevent.

DR. PERCY FRIDENBERG: The data as to the use of the speculum were taken from the classic treatises on the operations on the globe. They do not take into consideration the operation for squint, because in operations on the extra-ocular muscles there is no special danger, even if the patient should squeeze a little, as the globe has not been opened.

There are one or two other points that I should like to add. In regard to the operation for squint and tenotomies, there are some interesting observations by Hermann Knapp in his treatise in Norris and Oliver's "Textbook of Ophthalmology." He stated that penetration of the globe in the course of tenotomy comes from using scissors with needle points, which are unnecessary, and working from underneath, obliquely. Knapp stated that this subject came up in a meeting of the American Ophthalmological Society. H. Derby said he had had several patients in whom this had occurred; in his practice Knapp said he had had 1 or 2, and after that a number of the older members admitted to having observed several cases in their own practices in which this had occurred.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

TRANSLATED BY PERCY FRIDENBERG, M.D., New York

DOCENT DR. M. BÜCKLERS, Tübingen, *Reporter*

SECOND SCIENTIFIC SESSION

Monday, July 6, 1936, 3 p. m.

DR. W. MEISNER, COLOGNE, *Chairman*

IV. RÔLE OF THE OPHTHALMOLOGIST IN THE PREVENTION OF HEREDITARY DISEASES. DR. W. CLAUSEN, Halle.

(Continued from page 332)

Patients with cataract associated with myotonic dystrophy should, without exception, be sterilized. The procedure appears fully justified in myopia of extremely high degree, so-called excessive myopia, in which severe alterations are noted as a result of traction in the region of the posterior pole at a comparatively early stage, especially if these pathologic changes allow one to foresee an early or precocious visual invalidism and if, in addition to the myopia, there is a certain hereditary tendency to retinal detachment. In closing, the reader of the paper discussed the practical results of the sterilization laws and stated that he saw promise of far reaching elimination of hereditary ocular disease by the consistent and appropriate carrying out of a campaign of eugenic preventive hygiene.

DISCUSSION OF REPORT IV

HEREDITARY OPACITIES OF THE LENS. DR. M. BÜCKLERS, Tübingen.

Inheritance of lenticular changes has been known for a long time. This applies not only to the juvenile forms but to the senile as well, as has been demonstrated lately by the results of Vogt's studies of identical twins. The reader was able to follow a number of patients with senile cataract through three generations and got the impression that the same

form is perpetuated in a given family. Of 5,047 patients operated on for senile cataract in his service, 17.6 per cent stated that this condition had occurred in their family. Social and heredobiologic importance attaches first of all to those lenticular changes which are congenital or interfere seriously with vision at a very youthful age. The large group of zonular cataracts belongs in this category. Jess was quite right in calling attention to the fact that even the most experienced ophthalmologist meets, again and again, to his surprise, with forms of zonular cataract previously unknown to him. The extent of the variations is shown in a small selected series of observations from the reader's services in Berlin and Tübingen.

Familial distribution has been, so far, determined only for the form which may have some relation to coralliform cataract. Whether one is dealing in a given case of zonular cataract in practice with a variation in manifestation of an already familiar basic type or whether hereditary factors are concerned and what these factors may be are questions which at the present time can be answered in each individual case only by expert and intensive investigation of the family histories and genetic research. This study meets with great difficulties, as the cataract is generally extracted without its appearance having been noted or even sketched for record. Anamnestic data are practically useless for this purpose. The genealogies which we have collected generally concern families in which there was numerical heaping up of cases. If we wish to avoid the dangers of such a "selection on the basis of interestingness" (Just) we shall have to carry out a comprehensive heredobiologic census of the population. For this we need the cooperation of each and every ophthalmologist. Only then shall we have light thrown on the occurrence of apparently isolated cases. The fact that zonular cataract has been produced experimentally in animals in various ways, such as by toxic agents, massage and radiant energy, has given the impression that this disturbance may also be due to exogenous factors.

We know that Horner accepted rachitis, and Peters and his disciples, tetany, as the cause. Considering the enormous frequency of these two diseases (80 per cent) in our regions, it was always probable that a chance coincidence would be mistaken for a causal relation. But, as far as I know, here the pragmatic test, viz., examining children with a previous history of severe rachitis or of tetany for evidence of the presence of zonular cataract, has never been applied to this problem. At my suggestion, Dr. Gscheidel has for this reason started a survey of children formerly treated in the pediatric clinic. So far, only 40 patients have been examined, but it is interesting to note that of these not 1 showed the slightest trace of zonular cataract. As a general proposition the reader was inclined to agree with Waardenburg that, especially in regard to hereditary lenticular opacities, we cannot individualize and differentiate sufficiently. It is only by individualization and differentiation that with industrious and intensive research we shall finally get a clear conception of the nature of the multiple relations concerned in pathologic changes of the lens. The fact that we are able at all to recognize fine differentiating characteristics of cataracts today we owe primarily to slit lamp biomicroscopy and its founder, Alfred Vogt.

STERILIZATION OF SUBJECTS WITH CONGENITAL CATARACT. DR. JESS, Leipzig.

In their comments on this subject, Gütt, Rüdin and Ruttke said: "Preventive eugenics must be considered primarily in all families in which we note early and widespread occurrence of a cataract type. . . . Furthermore, those subjects should be sterilized who show, in addition to congenital cataract, other indications of physical or mental inferiority, such as cerebral disturbances and similar conditions. In these cases of congenital cataract accompanied with other 'complications' we know that operative procedures offer a very unsatisfactory prognosis as to ultimate vision . . . At the very least, all subjects with so-called complicated congenital cataract should be rendered incapable of reproducing." Fleischer was then cited to the effect that a good visual result is generally not to be arrived at even with a correctly performed and uneventful operation for congenital cataract. This author then went on to say: "From this welcome point of view, in accordance with which the decisions as to sterilization must be made," etc. In accord with this dictum, as experience in a large number of cases has demonstrated to the reader, many practitioners have been convinced that the law requires the patient with any form of cataract recognized as definitely congenital to be sterilized without any further consideration. Certainly, every case of cataract recognized as congenital and hereditary must be reported to the district health officer. However, the decision as to sterilization concerns the judicial body (*Erbgesundheitsgericht*), which in each case bases its decision on the careful and detailed reports of the expert medical examiners. It need hardly be added that sterilization is to be advised without hesitation in the aforementioned cases of "complicated" congenital cataract. However, the reader definitely denied the validity of Fleischer's dictum that in all cases of congenital cataracts we must expect an unsatisfactory visual result from operative intervention. He referred to a recent publication of Heine (of Kiel) reporting the subsequent life history of subjects with congenital cataract, in which the author pointed out definitely how many persons are found to be quite capable of following various trades or professions after a correctly performed operation and how important it is to consider and judge each case of congenital cataract individually, with the greatest care and with regard for all the conditions. The reader's own experience tallied with that of Heine, and personal communications from Clausen, Fleischer and others have convinced him that a general consensus has been reached and that sterilization is not to be advised in many cases of zonular cataract, especially in cases of small opacities of limited extent which do not markedly interfere with sight and in which complications are entirely lacking, even if heredity has been proved.

The responsible adviser of the court will always have to ask himself to consider seriously the question whether in such cases families might not be extirpated who, after correction of a comparatively slight lesion, could be of service to their country. We must bear in mind that it is not wise to go beyond the limit in cutting down quantity in favor of quality.

RAPIDLY PROGRESSIVE PIGMENTARY DEGENERATION OF THE RETINA. DR. STOCK, Tübingen.

A man of 25 was perfectly healthy until one and a half years previously. Within this space of time he became hemeralopic; then he showed a concentric contraction of the visual field, which progressed rapidly. The patient became blind six weeks before death. It was not the macula but an eccentrically located area in the temporal field which preserved its function longest. Clinically the patient presented the picture of retinitis pigmentosa, except that the pathologic changes were much grosser than usual. At first glance the fundus picture appeared to be that of an old, severe choroiditis.

However, with the binocular opththalmoscope and in the upright image it could be definitely noted that the foci had been produced by partial disappearance of pigment, partly by its heaping up in irregular clusters (*Klumpen*) and also by its emigration into the retina. While the patient was under clinical observation, extremely minute hemorrhages occurred in the layer of rods and cones and in the pigment epithelium, which later disappeared.

Histologically the process was one of decay of all the retinal nervous elements, beginning with the rods and cones, but almost at the same time involving the bipolar and ganglion cell layer in destruction. This degenerative process took place so rapidly that there was hardly time for proliferation of glial or connective tissue, so that vacuoles filled with fluid formed in the substance of the retina. The optic nerve still showed fairly good staining of the medullary sheath. In the brain and spinal cord almost all the ganglion cells, especially those of the cortex, had practically disappeared or were in the course of degeneration. In the meninges there were a few minute hemorrhages. Round cells were observed encircling some vessels of the brain and of the eye. As the patient was otherwise perfectly well and the postmortem examination failed to reveal any pathologic lesions in the viscera, it is impossible to venture even a surmise as to the cause of this acute degeneration of the nervous elements of the eye and central nervous system.

DR. BARTELS, Dortmund: Judging from the literature of our times and also from the discussions and opinions expressed at this meeting, one gets the impression that nowadays we do not consider only the disease in deciding our question but also what the patient can accomplish with or in spite of his disease. The law instructs that children whose vision does not permit them to follow the course of instruction at the ordinary schools and who have to be referred to a school for the blind are to be considered as sightless and if they are the subjects of hereditary disease mut be sterilized. But here, too, we have to take into consideration the feeblesighted. The schools for this class, it must be remembered, owe their origin directly to the fact that children were sent to schools for the blind when they were not "blind" at all, but merely feeblesighted. However, they are quite capable of following the entire school course if special methods are employed. Accordingly, they cannot be considered as blind. Those feeblesighted children who have a hereditary condition do not need to be reported without any further consideration, but should be reported only in case it appears that this condition has already been noted to be in the ascendancy. It might be well to take notice of what can be accomplished in a family

with congenital cataract. Incidentally, we note that in the schools for the feeblesighted there are many subjects with extremely high degrees of myopia and that just these children are unusually intelligent. The reader protested against the sterilization of male subjects with Leber's atrophy of the optic nerve on the ground of its being unnecessary. These patients never have diseased offspring. It is a great rarity for women to be affected with this disease, and those who have it can be sterilized. It is advisable, in order to avoid mistakes, to have each patient with such a condition examined independently by two ophthalmologists. Furthermore, the reader insisted that the judge of final instance, or the referee (*Oberbegutachter*) should render his decision not only on the basis of the documents in the case but after personal examination as well.

DR. LÖHLEIN, Berlin: There is no law which so well deserves to be called a humane statute, if judged by its meaning and purpose, as the law to prevent hereditarily diseased progeny. For it intends to prevent and to spare the individual, the family and the people as a whole a flood of the greatest sorrow by taking preventive action in advance against the destructive action of hereditary disease. It is true that the right, which, in agreement with the traditional wish of the medical profession, this law places in the hands of the physician, carries with it a great and serious responsibility, and we cannot subscribe to the opinion that the mere diagnosis of a hereditary malady alone would suffice for a decision to sterilize. On the contrary, the law requires also, as far as ophthalmology is concerned, that a condition of hereditary blindness or of serious developmental defect of the eye be present. The reader expressed the conviction that his colleagues all feel, as he does, the great responsibility of the ophthalmologist as an expert and that they follow the same principle, viz., limitation of their decision to unequivocal cases in which the necessity for the procedure can be made evident to the comprehension of an intelligent layman. We all have the standpoint that it is better at times to do too little than at any time to recommend sterilization lightly. Besides, it is the principle of the reader, as it surely is that of other conscientious experts, always to enlighten the patient and to convince him to the point where he himself, as far as possible, makes the request for sterilization and does not retain the feeling that he has been subjected to the coercion of a strict law. He will then consider his resolution, however hard it was for him to make, as his own liberating act. When we have spread the understanding of these requirements, step by step, in ever wider circles we may also hope to carry out successfully a task which is just as important, namely, in our capacity as advising physicians to have our purely humane counsel as to the self-denial of progeny followed in those cases which do not come under the provision of the law and in which a serious hereditary condition gives reason to anticipate danger but in which there is no actual blindness. The reader had in mind families with summation of cases of high myopia and detachment; the sisters of patients with Leber's disease, and similar cases. He stated that he was not in accord with the view that we put too high an estimate on what an individual can accomplish with energy and will, even if his visual remnant is, if taken by itself, insufficient. If he is the carrier of a serious hereditary malady and has a defect of vision which would

mark the average individual as practically blind, he should draw the logical conclusion, for it is hardly to be expected that the same unusual energy and will-power in his descendants will help them over the same serious defect. Several colleagues here have expressed a wish to have short directions drawn up and worked out which the ophthalmologist could follow in giving his expert findings and individual recommendation. The reader thinks it would not be right to have printed regulations planned which would lead the physician engaged in a case to look up the requirements in a perfunctory way and give his decision offhand as a sort of rubber stamp. The problem is one which is to be decided in each case, individually, and carries great responsibility with it. It takes time and labor. But one is dealing with matters of such unusual moment and serious importance that the expert should not be put in the position of having his well based, carefully considered and proved point of view superseded by any purely technical rules and regulations. We naturally aim to exchange the data of our experience and observations and so give facility to the judging attitude and uniformity to the method of arriving at a decision for each consultant. A valuable service is being rendered to such physicians in our broad, thorough and intensive discussions here of the question in its widest bearings, by the new edition of the commentary on the sterilization law, and by the publication of a handbook of hereditary disease, which is being prepared. It is important that we as experts should clarify the points in each individual case thoroughly, and, having done so, we should strongly urge that our expert judgment should receive full and due consideration. The reader is convinced that the difficulties which in this respect were at times presented in the past from the juristic side would tend to disappear as the jurist gained insight into the complexity of the conditions as to which the expert has to testify and as to which he alone can decide.

Dr. Meesmann, Kiel: Allusion was made to papers to be presented within the following days of the meeting by Holtz and Meesmann, which also deal with the subject of zonular cataract. It is improbable that rachitis, per se, leads to zonular cataract. There is required, in addition, some lowered function of the epithelial bodies. As this condition can be effectively treated today, according to Holtz, it is important to establish contacts with families in which zonular cataract is common, so that the women, particularly, can be treated during pregnancy with an ergosterol preparation.

Dr. von Bahr, Upsala, Sweden: Both Bücklers and Meesmann reject rachitis as a cause of zonular cataract. The reader thinks this is incorrect, as is Bückler's rejection of tetany as the etiologic factor. There are, as a matter of fact, cases of zonular cataract in which the condition is hereditary, and the reader has collected from the literature records of upward of 30 families showing a dominant heredity. But of over 700 patients with zonular cataract, well over half had suffered from cramps in childhood. This could not have been coincidence. Many showed signs of rachitis, too. J. Kugelberg, in Upsala, collected the data in a follow-up of about 150 children who had been treated at the hospital for spasmophilia in infancy or childhood. Of these, about one third had lenticular opacities similar to those observed in cases of zonular cataract, such as isolated peripheral spokes *(Reiterchen)*, while

in three cases there was a fully developed zonular cataract. After presenting these clinical observations the reader submitted the data of some of his own experimental research. One hundred and nine young rats in 23 series were fed a rachitogenic diet, and the presence of rachitis was established by the x-ray plates. Forty-four of these rats received the diet mentioned uninterruptedly. They did not show cataract. For the other 65 rachitic rats the diet was modified so that it contained more phosphates. Of these 65 rats, 48 showed lenticular opacities. The rachitic rats reacted to the change in diet with an increase of response to irritation by the galvanic current and in a number of cases with spontaneous cramps or tremor. The controls, which had been given vitamin D in addition to the special diet, showed a very slight excess of irritability but no cataract. Lenticular opacities appeared in the eyes of the rachitic rats following excess irritability, in the majority of cases within forty-eight hours, and in many, in fact, within twenty-four hours, after the change in diet. These opacities were at first subcapsular, but with the development of the lens they had the appearance of zonular cataract. This condition, then, is due not to rickets alone but to rachitic tetany, or spasmophilia.

DR. STOCK, Tübingen: In his report, Clausen stated that we are all agreed that neuro-epithelioma (glioma) of the retina belongs, under all circumstances, to the hereditary diseases, and the patients are, accordingly, subject to sterilization. Personally, I disagree. In 28 cases of glioma in patients operated on in our Tübingen clinic in the last year or two we have had careful studies instituted and in only a single case found that an eye had been lost in the ascendancy. And even in this case it was not definitely determined whether the loss of the eye was due to glioma. After all, we do not sterilize in cases of carcinoma or sarcoma, although they might count as hereditary affections as logically—or as illogically!

DR. FLEISCHER, Erlangen: The reader declared himself generally in accord with the preceding speakers, especially with Clausen in his reference to the serious responsibility which a physician takes on himself when he makes the report to the official medical examiner, as required by law, of a case of hereditary disease or certifies as to such a condition before the tribunal concerned with genetic hygiene. However desirable, hard and fast rules of invariable and permanent application cannot be laid down in the present state of knowledge, either for notification or for the certification just mentioned, but only general suggestions and directions can be given. Each subject must be conscientiously examined and tested. On the other hand, the reporting of suspicious cases cannot be avoided if one considers one's duty to the law and the community. In just such cases it is the notification which brings the matter before a duly constituted board for thorough investigation of the family history. It goes without saying that tact rather than drastic methods should characterize the activities of the physician, the official medical examiner and the juristic board.

DR. HERTEL, Leipzig: As was already mentioned by Bartels, difficulties are raised in these matters by the fact that the term blindness, which the law demands as a prerequisite for sterilization, has not been accurately defined. In the courts, statutes, codes for insurance and / or workmen's compensation laws there are many different specifications of

blindness and, especially, of the limits of useful vision. This uncertainty makes it very difficult to decide whether a patient is to be reported for sterilization and may be used by the patient himself to raise objections. This difficulty is most marked if the hereditary condition is monocular. We have noted large unilateral colobomas, aniridia, anophthalmos, macular degeneration and so on as evident signs that the subject is the carrier of pathologic heritage and that, accordingly, sterilization should be demanded. But what are we to do if the fellow eye is perfectly sound and has good vision? The carrier is perfectly capable of carrying on a trade, and we would, in a way, be acting against rather, than in accordance with, the law if we were to cause his sterilization. If the visual function is to be considered as a factor in the law and its application, we must ask the authorities to give us a more exact definition of this concept.

DR. COMBERG, Rostock: The reader has gained the impression that today's statements are of importance, as they complete and confirm what has been set down in the literature on the subject in the last year or two. It would be desirable to have some sort of standing medical board in Germany, composed of individual ophthalmologists, whose office it would be to test out the various questions and moot points continuously and currently, either in lectures or / and correspondence, so that a consensus might be obtained as to the large, directional ideas which would be competent in deciding in the individual case. It is necessary for the large majority of ophthalmologists who do not take part in the proceedings of the German Ophthalmological Society to have a more detailed and definite basis for determination and judgment in difficult cases.

DR. MÜGGE, Eisleben: The question whether we are to sterilize or not undoubtedly confronts the practical ophthalmologist with a serious responsibility, and I do not wish to be understood as pleading in favor of exaggerated claims and suggestions for sterilization. But, in regard to glioma, I have been greatly surprised to hear the view expressed here today repeatedly that in the majority of cases the condition is not hereditary. In some of the cases it undoubtedly is hereditary, and this applies also to the 2 brothers mentioned by Clausen in his report and who were my patients. The misery which these 2 children brought on their family may be judged by the facts which I add here. Within the space of a year and a half, one of the brothers had to have both eyes, the other, one eye, enucleated. In both subjects exenteration of the orbit had to be performed, in spite of which finally there was extension to the other eye, requiring enucleation. Any one who has lived through an experience of this sort will rather advise sterilization in one case too many than in one case too few.

(To be continued)

Book Reviews

Ocular Therapeutics. By Sanford R. Gifford, M.D. Second edition. Price, $3.25. Pp. 341, with 60 illustrations. Philadelphia: Lea & Febiger, 1937.

The first edition of this book appeared in 1932 and contained 272 pages and 36 illustrations. The new edition has 69 more pages and 24 more illustrations than its predecessor. The headings of the chapters are unchanged, but a new chapter on diseases of the orbit has been added. The format is unchanged except that the author has used italics for directing attention to matters of fundamental importance in therapy. This is a definite asset.

Much new material (based on the work of Bellows) concerning the relative efficacy of cocaine, phenacaine hydrochloride, butyn, nupercaine, pontocaine (paranormobutylaminobenzoyldimethylamino-ethanol hydrochloride) and metycaine has been added to the chapter on anesthetics. Various methods of infiltration anesthesia are discussed more fully than in the earlier edition. Vitamins, which were scarcely mentioned in the first edition, are given 5 pages; thirty drugs have been added to the list of drugs most used in ophthalmology, and a convenient procedure for preparing buffered ophthalmic solutions is outlined briefly. One correction should be noted at this time. In the formula for Acid Buffer Solution No. 1, at the top of page 62, 12.4 Gm. of boric acid is to be used instead of 6.2 Gm. New material concerning diathermy and phototherapy has been added to the chapter on physical therapy.

The chapter on diseases of the cornea has new sections on the treatment of marginal ulcer, Mooren's ulcer, smallpox keratitis, superficial punctate keratitis, epithelial dystrophy, neuroparalytic keratitis and keratitis due to lagophthalmos.

The new chapter on diseases of the orbit is concerned with orbital cellulitis; thrombosis of the cavernous sinus; exophthalmos in association with hyperthyroidism; orbital hematoma; fracture of the wall of the orbit, and pulsating exophthalmos.

This new edition of "Ocular Therapeutics" is a sane, well balanced and conservative presentation of the various drugs, physical agents and mechanical procedures which the author has found to be of value. It deserves to be on the bookshelf of every ophthalmologist, and it should be required reading for residents in ophthalmic hospitals.

W. F DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

Ophthalmological Society of Egypt

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3, Midan Soliman Pasha, Cairo.
Time: March 1938.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City. Time: May 24-27, 1937.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501 Seventh St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221 Fifth St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514 Sixth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.